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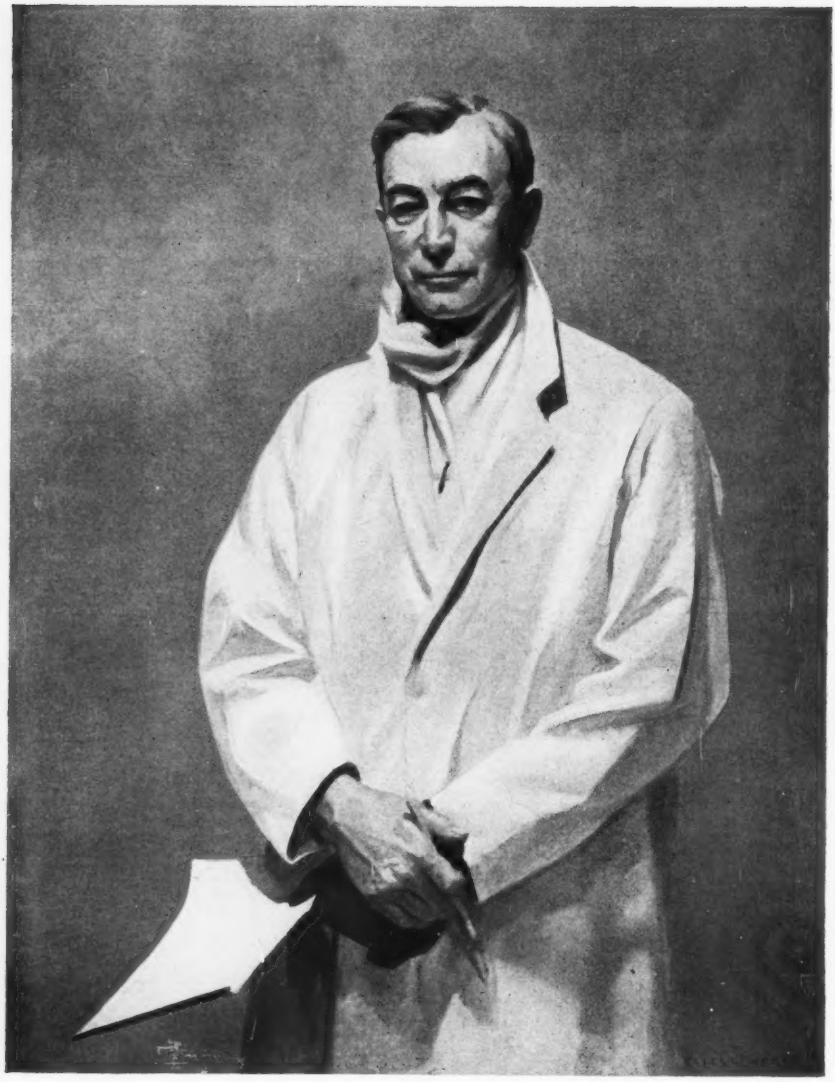
JOINT MEETING OF THE AMERICAN, BRITISH, AND CANADIAN ORTHOPAEDIC ASSOCIATIONS IN QUEBEC

Twenty years ago a joint meeting of the British and American Orthopaedic Associations was held in London. The occasion was a happy reunion of surgeons who worked together during the First World War under the leadership of Sir Robert Jones. Hey Groves, who was president of the British Orthopaedic Association, opened a discussion on the treatment of fractures of the neck of the femur. Fred Albee, president of the American Orthopaedic Association, displayed the skill of his carpentry in bone grafting. Royal Whitman used every device of charm and determination in resisting yet another demand for statistical evidence by which to support his claims for the abduction plaster spica. Smith-Petersen demonstrated a post-mortem specimen from which every member of the audience tried unsuccessfully to pull out the first three-flanged nail they had seen. Joel Goldthwait discussed postural deformities of the spine. Putti gave a polished presentation on the subject of congenital dislocation of the hip. Sir Arthur Keith showed specimens in the Museum of the Royal College of Surgeons of England. Steindler performed his muscle slide operation for pes cavus. Winnett Orr was developing the closed plaster treatment of bone infections; Bohler was advocating the Davis method of reducing fractures of the spine; Watson-Jones was drawing diagrams of a patient suspended between two tables.

After this interval of years it is easy to recognise the signs of progress which was imminent. Much water has since flowed beneath the bridges of the Thames. As A. P. Herbert would point out, the river is tidal and much of it has flowed back again. The Whitman plaster is no longer the treatment of choice for fractures of the femoral neck. Inlay bone grafting has been superseded by other methods of transplantation. Steindler's operation for pes cavus has been abandoned. Postural reduction of fractures of the spine is being discredited. The closed plaster treatment of bone infections is seldom indicated. Surgical pioneers whose work has been supplanted must remind themselves of the words of Priorov of Moscow: "It is poor tribute to a surgeon if in his lifetime he is not excelled by his own pupils."

That there has been striking and important progress cannot be denied, and it is perhaps surprising that no other joint meeting has been held in twenty years to celebrate the discovery in Britain of penicillin, and the development in the United States of safe methods of internal fixation—discoveries and developments which have revolutionised the treatment of bone infections and bone injuries. We recall the resolution of the American Orthopaedic Association, assembled in London, "expressing its appreciation for gracious hospitality and lavish expression of good fellowship. Closely bound to our British colleagues by every kind of human tie we express the hope that this delightful meeting may be the forerunner of other similar happy reunions."

Much of the credit for arranging the second joint meeting of the two Associations, now to be joined by the Canadian Orthopaedic Association, is due to Robert I. Harris of Toronto, who this year is president of the American Orthopaedic Association. So far as it is possible for him alone to assure success, it *is* assured by his American allegiance, Canadian nationality, and British instinct. So far as it is possible for the American Orthopaedic Association to assure success, it *is* assured by the fact that they have elected as president, vice-president, and chairman of the programme committee, three surgeons, none of whom is American—Robert Harris of Toronto, Alberto Inclan of Havana, Cuba, and John McDonald of Toronto.



Robert I. Harris of Toronto,
President of the American Orthopaedic Association.

The American and Canadian Associations will be represented fully at this meeting, and if the decision had rested solely with the orthopaedic surgeons of this country the British Orthopaedic Association would have been represented no less fully. Over eighty fellows and members planned the journey to Quebec, many arranging to be accompanied by their wives. In due course they were informed that limited financial resources made it necessary for them to travel alone. Now it is learned that they themselves may not travel. No more than token representation is possible, by a very limited number of members of the Association. Other members who, we know, are resenting the decision forced upon us, must remind themselves of the words of Lord Keynes, uttered in the House of Lords as long ago as May 1944:

" We shall emerge from this war having won a more solid victory over our enemies, a more enduring friendship from our allies, and a deeper respect from the world, than at any time in our history. This is because we have sacrificed every precaution for the future with

S. Alan Malkin of Nottingham, President of the British Orthopaedic Association.

J. Edouard Samson of Montreal, President of the Canadian Orthopaedic Association.

a fanatical single-mindedness which has had few parallels. But the full price of this has still to be paid. In thus waging the war without counting the cost we—and we alone of the United Nations—have burdened ourselves with a weight of deferred indebtedness to other countries beneath which we shall stagger. We have already given to the common cause more than all we can afford."

It is true that we are staggering. It may be true that we have given more than all we can afford. But we shall survive. We shall indeed survive. On this occasion our representation is no more than a token, the greater part of which is made possible by the enduring friendship of our allies in the American continent whose offer of hospitality to thirteen young ambassadors of this country is characteristic of their generosity. But already we are laying plans for the next joint meeting in London, to include not only the Orthopaedic Associations of America, Canada, and Britain, but also the Orthopaedic Association of Australia, and, we trust, the Associations yet to be formed in Africa, New Zealand, India, Pakistan, Ceylon, and the British Colonies. In the words of Franklin Delano Roosevelt, whose memorial in Grosvenor Square was unveiled recently: " The only limit to our realisation of to-morrow will be our doubts of to-day. Let us move forward with strong and active faith." EDITOR.

PARAPLEGIA IN CERVICAL SPINE INJURIES

Thoughtful and comprehensive studies of spinal injuries which were published by some of our surgical forerunners in the great text-books, monographs, and journals of the last century, established four possible methods of displacement—flexion, extension, lateral dislocation, and rotation. It was established that excessive flexion was the most usual means of cervical dislocation—excessive flexion combined at the same time with great pressure on the head; and twenty years ago this writer reviewed and brought up to date the broad principles underlying this mechanism (Proceedings of the Royal Society of Medicine, 1928, 625). At the same time special consideration was given to the most usual sites of injury. It seemed that fractures, or in the cervical region dislocations, occurred at the points of maximal curvature under stress. Since the spinal regions of greatest mobility were found to coincide with the greatest liability to injury, these were designated the " critical points of spinal injury " —a new proposition, because until 1928 it had been accepted that the spine broke at the junction of a mobile and a relatively immobile region. It would be wrong to assume that relative immobility of the thoracic spine had no effect whatever in determining the site of injury. What had been overlooked was that mobility of the adjoining portion did not in itself constitute the factor requisite for injury. There was some quality that needed taking into account in the mobility of the cervical spine and this, I thought, was its ability to produce a curve, the injury happening as it does with any material under bending-strain at the apex of the arc of curvature. This I still believe to be true, though I have been content to state the principle without elaborating it.

In the paper referred to, injury by extension was mentioned but its mechanism was not developed. That development has had to wait until to-day. In this issue of the Journal are two papers on extension injuries of the cervical spine which have a significance of conception that is their own. This does not lie in the fact that violent over-extension of the head can produce serious injury, but in the deductions drawn by the authors from their observations. Description of extension injuries can be found from the days of Malgaigne onwards; for example in 1853 Butcher reported an extension injury with rupture of the anterior common ligament. However, little serious thinking had been applied to the extension mechanism, except perhaps by Wagner and Stolper. These authors illustrated a good specimen of rupture of the anterior common ligament at the second-third cervical level which caused death from high spinal injury (Die Verletzungen d. Wirbelsaule 1898. Fig. 91). Significantly, the specimen showed little or no displacement and some who described similar cases made the same observation, though none drew the inferences now made by Barnes, Taylor, and Blackwood. Wagner and Stolper, in an analysis of 205 cases collected from the Journals, found that 141 were flexion injuries, forty-nine were rotational injuries, and only fifteen were extension injuries; the high proportion of rotational injuries being accounted for, no doubt, by the inclusion of lesions of the atlas and axis. The proportion of extension injuries in the series reported in this issue by Barnes is much greater, though, as he says, there may be special reasons. But it could be true that they are more common than we have imagined because our minds have been occupied by a satisfactory explanation and we have failed to inquire closely enough into the possibility of the head being extended rather than flexed by the force of injury. It is difficult to know in what direction force has been applied to the head. When there is forward displacement the question scarcely arises. But when there is no displacement we are invited to believe that the injury has probably been by over-extension. For my part I am ready to accept this as correct for most cases. It is probably not absolutely true always. The point is not one to be laboured, for the difficulty of knowing the true facts of any injury in terms that would even begin to satisfy a physicist is too well known to ourselves. Our method of reasoning has usually to be deductive rather than inductive, but that does not necessarily mean that our conclusions are without value.

The special point about the contribution of Barnes is the significance he attaches to age; that extension injury is more common in those with relatively rigid necks and naturally therefore in the later decades of life. This is a first-rate piece of observation, supported by Taylor and Blackwood, for in such circumstances new mechanics must obviously apply.

The suggestion is made that in the majority of cases where there is cord injury up to a level, but with no evidence of displacement, the injury has usually been occasioned in this manner, by hyper-extension. With the exceptions drawn from very gross flexion joint ruptures it seems that they have gone far towards proving their point. If this conclusion is substantiated by other observers, and it probably will be, we have found an answer to a problem which has puzzled surgeon and neurologist alike for over a century. A virtue of this clarification will be the disposal of spinal concussion, a state of which some have written as if it could occur without anything coming into contact with the cord—an absurd notion indeed.

The part played by extrusion of disc tissue in causing damage of the cord has come to the forefront in recent years. So long ago as 1909 Thorburn wrote " more rarely the damage is done . . . from the forcing back of intervertebral cartilage which forms a projecting shelf pressing against the cord " (Burghard's " System of Operative Surgery "). It would be untrue to suggest that Thorburn considered this to be a really significant feature of spinal trauma. He observed it, as had many others, at necropsy but regarded it as scarcely more than part of the disturbance of normal relationships produced by great violence. He was unable to illustrate it as an isolated event and proposed no particular treatment. In recent times the present writer has considered it a more serious feature (Proceedings of the Royal Society of Medicine, 1940, 651) and a discussion a few weeks ago in London is reported in detail in this number of the Journal. Beyond doubt further observations will put it in proper perspective.

The real problem of spinal injury is not altered materially by change of opinion as to what the agent of injury may be. If the cord has been badly damaged it is still fruitless to operate. If laminectomy alone had been the right step then older surgeons, who did this operation in cases of injury much more often than we do to-day, should have had far more success than, in fact, they had. But Barnes is quite right in pointing out that total extinction of cord function can occur without haemorrhage into the cord, a fact with which this writer has long been impressed (British Medical Journal, 1936, 1125). Unhappily, it does not follow that recovery is possible, for disruption of neural elements can occur without bleeding. The cord is not so vascular a structure that it cannot be damaged bluntly without haemorrhage.

The two papers published in this number have the virtue of that brevity and clarity with which new ideas are best expressed. GEOFFREY JEFFERSON.

TRAUMATIC URAEMIA

" More lives have been lost than ever were saved by the use of the tourniquet." Why does the prolonged application of a tourniquet cause death from uraemia? Is this complication the same as the uraemia of the crush syndrome? Is it safe to believe that in such cases life can be saved by amputation? What is the relationship between crush and tourniquet anuria, the anuria of mismatched blood transfusion and blackwater fever, and the uraemia of surgical shock?

In our last number we reviewed the work of Trueta, Barclay, Franklin, Daniel, and Pritchard who, in " one of the most remarkable researches in recent years," showed that there is a dual circulation in the kidney under sympathetic control which can be stimulated by arterial injury in the lower limb. The relationship between the anuria of cortical renal ischaemia due to lower limb injury, that of renal tubule blockage by the products of mismatched transfusion, and of renal anoxia due to fluid loss and shock, are not yet fully elucidated. In this number of the Journal we have invited from Dr Darmady a critical review on the subject of traumatic uraemia, and in a forthcoming number of the Journal we hope to publish a contribution on orthopaedic applications of the recent researches in Oxford.

EDITOR.

PARAPLEGIA IN CERVICAL SPINE INJURIES

ROLAND BARNES, GLASGOW, SCOTLAND

From the Department of Orthopaedic Surgery, University of Glasgow

The first recorded reference to paraplegia in cervical spine injuries is to be found in the Edwin Smith papyrus, written some four thousand years ago. The unknown author comments briefly upon the condition: "One having a crushed vertebra of his neck, he is unconscious of his two arms and two legs, and is speechless. An ailment not to be treated." Such pessimism was not without justification for in spite of notable contributions by Walton and Taylor, and more recently the introduction of caliper traction by Coleman, McKenzie, and Crutchfield, considerably more than half the patients with paraplegia die from the complications of spinal cord injury, and many survivors are left with varying degrees of paralysis.

One of the most puzzling features of injuries of the cervical spine is the lack of correlation between the degree of vertebral displacement and the severity of the spinal cord lesion. There are cases with no radiographic evidence of bone injury in which the cord is irretrievably damaged; others, with gross dislocation, may have no paraplegia. It is commonly believed that the spinal cord is damaged by the dislocated neural arches, and in the absence of radiographic evidence of bone injury it is assumed that spontaneous reduction of the dislocation has taken place. This view is not, however, in accord with experimental or post-mortem observations.

Radiographic study of specimens in which the dural sac has been filled with lipiodol shows quite clearly that the dislocated neural arches do not produce a degree of narrowing of the spinal canal which would compress the cord until there is locking of at least one articular process (Fig. 1). Once the articular processes are locked the dislocation is usually stable, and reduction cannot be secured without manipulation. Furthermore, examination of post-mortem material and observations at operation have convinced me that spinal cord injury can occur in the absence of any vertebral dislocation; and unless there is clear radiographic evidence of fracture of the articular processes which will permit spontaneous reduction of the dislocation we should look for an alternative explanation of the cord injury.

TYPES OF CERVICAL INJURY

Damage of the cervical cord may be caused by flexion injuries and hyperextension injuries. The number of cases of each type of cervical injury in a series of twenty-two patients with paraplegia is shown in Table I.

TABLE I

Flexion injuries—	
Dislocation	8 cases
Crush fracture of vertebral body	4 ,,
Acute retropulsion of intervertebral disc	3 ,,
Total	15 ,,
Hyperextension injuries—	
Dislocation	1 ,,
Injury to arthritic spine	6 ,,
Total	7 ,,

Three types of flexion injury may be recognised: 1) anterior dislocation; 2) crush fracture of a vertebral body; 3) acute retropulsion of an intervertebral disc. There are two types of hyperextension injury: 1) posterior dislocation; 2) injury to an arthritic spine.

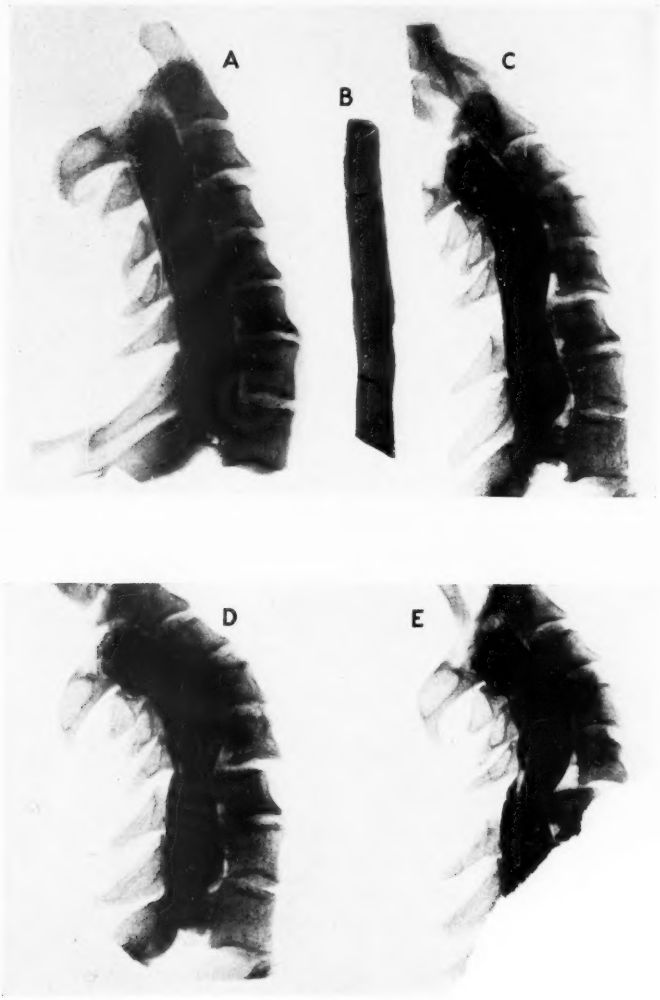


FIG. 1

Experimental displacements of the cervical sp
been filled with lipiodol. A shows the specime
show degrees of flexion dislocation. B is a sof
cord for comparison of size. In C there is subl
In D one articular process is locked. In E b
The spinal cord is not compressed by the di
one articular proces

It must be emphasised that these twenty-tv
the reason that some were treated in a Spinal In
obscure types of injury. It is likely therefore th
proportion of dislocations. The age incidence
All flexion injuries, and the one dislocation in e
of age; no hyperextension injuries in arthriti

fifty-seven years of age. The mortality rate in all types was high except in the group of three patients with disc injury, all of whom survived.

FLEXION INJURIES OF THE CERVICAL SPINE

Cervical dislocations—In dislocation of the cervical spine it is generally agreed that cord injury may be due to a combination of two factors: *a*) pressure on the dorsal surface of the cord by the dislocated neural arches; and *b*) often co-incident compression of the ventral surface of the cord by retropulsed disc material. Skeletal traction by means of a skull caliper is the treatment of choice. The dislocation can be reduced easily, without risk of further damage to the cord, and with better prospect of relieving the pressure of the disc than by other methods. The nine dislocations in this series presented no unusual features and it would be unprofitable to discuss them in further detail.

Acute retropulsion of disc—There were three cases of this type, all due to forcible flexion of the head on the trunk. In none was there radiographic evidence of bone injury but all the radiographs showed narrowing of one intervertebral disc.

Case 1. *W. T., aged* 34 *years*—Fell over the handlebars of his cycle on to the back of his head. He was examined within an hour of the accident and found to have incomplete paralysis and sensory loss in the lower limbs, trunk, and upper limbs. Radiographic examination showed lipping of the anterior margin of the third and fourth cervical vertebrae and narrowing of the intervertebral disc (Fig. 2). There was no bone injury. On the day after injury paralysis of the legs had increased and there was retention of urine. The Queckenstedt test showed complete spinal block, and myelography revealed an arrest of lipiodol opposite the damaged intervertebral disc (Fig. 3). By the third day there was complete paralysis of both legs. Laminectomy was performed, and a large protrusion of the disc was observed between the third and fourth vertebrae. The cord was compressed between the disc and the laminae, ·d pulsation did not return to the cord until the third, fourth, and fifth laminae had been removed. ·e was steady recovery of motor power and sensation after operation, and two months later the patient ·e to walk without support.

-Radiographic evidence indicated that the disc was degenerated before the ·his no doubt predisposed to " massive " protrusion of disc substance into the ·esult of flexion injury.

·e cases, considerable recovery of paralysis occurred without operative ·d there is therefore no proof that the cord injury was caused by ·Nevertheless the mechanism of injury, the narrowed inter- ·e of radiographic evidence of bone injury, make the diagnosis

·eported by Brooke (1944) and Brown and Little (1939). ·in Brooke's patient gave normal readings, post-mortem ·sion of the disc which was causing severe compression

·he treatment most likely to relieve pressure on the ·ing that the disc is not degenerated before injury, ·isc height, and it is possible that some at least of · into the intervertebral space. Even if this does the knuckle pressing on the cord (Jefferson 1940). ·stedt test gives normal readings of cerebro-spinal ·der severe compression and preliminary trial of ecovery of paraplegia within a few days of the ·ould be considered even when the Queckenstedt ·and Brooke (1944) have reported cases of cord manometric readings.

FIG. 2

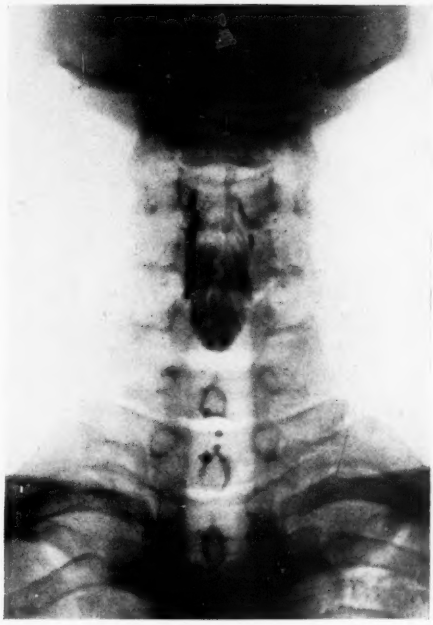


FIG. 3

Case 1. W. T., aged 34 years. Flexion injury of the cervical spine with incomplete paraplegia. Fig. 2 shows that the intervertebral disc between the third and fourth cervical vertebrae is narrowed but there is no bone injury. Myelography after injection of lipiodol into the lumbar theca (Fig. 3) shows complete block at the level of the herniated disc.

FIG. 4

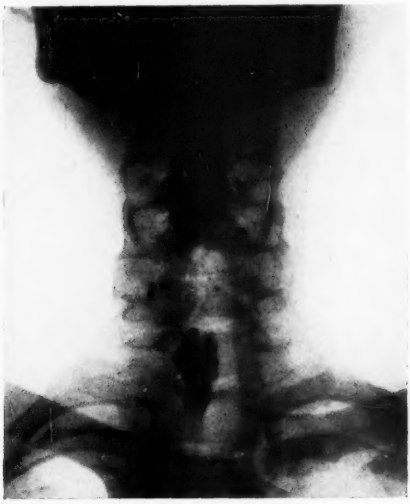


FIG. 5

Case 4. A. G., aged 30 years. Fracture of cervical spine with residual paralysis of upper and lower limbs two years after injury. Fig. 4 shows crush fracture of sixth cervical vertebra. Fig. 5 shows the myelogram; there is a filling defect opposite the fractured vertebra.

TABLE II

POSTERIOR PROTRUSION OF INTERVERTEBRAL DISC

Case	Age	Nature of injury	Radiographic appearances	Type of paraplegia	Queckenstedt test	Operative treatment	Result
1 W. T.	34	Fell over handlebars of cycle on to back of head	Narrowing of disc between C.3 and 4 with arthritic lipping of adjacent vertebrae. No bone injury. Myelograph revealed complete block at the above level	Incomplete	Complete block	Laminectomy	Considerable recovery
2 W. S.	39	Fell from cycle and turned a somersault	Marked narrowing of disc between C.5 and 6 with arthritic lipping of adjacent vertebrae. No bone injury	Incomplete	No block	Nil	Almost complete recovery
3 B. R.	40	Fell headlong downstairs	Slight narrowing of disc between C.3 and 4. No other injury	Incomplete. Brown-Séquard syndrome	No block	Nil	Considerable recovery

TABLE III

COMPRESSION FRACTURES

Case	Age	Nature of injury	Radiographic appearances	Type of paraplegia	Treatment	Result
4 A. G.	30	Lorry collided with another vehicle. Thrown on to back of head	Compression fracture of C.6. Myelograph later showed persistent anterior filling defect at level of fracture	Incomplete	Plaster jacket, three and a half months	Incomplete recovery
5 J. T.	40	Fell twenty feet on to deck of ship	Comminuted fracture body of C.5	Complete	Nil	Death on the day of injury. Post-mortem—herniation of disc in addition to fracture; severe crush of cord
6 W. McA.	18	Performing hand spring; arms gave way and fell heavily on neck	Compression fracture of C.5.	Complete	Nil	Death second day. Post-mortem — no evidence of dislocation; severe crushing of cord at level of fracture
7 J. R.	18	Root of tree fell on to back of head	Compression fracture of C.4 and 5	Incomplete Queckenstedt—no block	Caliper traction ten weeks	Incomplete recovery

When there is strong presumptive evidence of disc protrusion, and spinal block is demonstrated by manometry, decompression of the cord is indicated urgently. In these circumstances laminectomy is likely to be more effective in relieving pressure on the cord than caliper traction.

Crush fracture of cervical vertebra—Four patients sustained crush fractures of the cervical column without radiographic evidence of dislocation of the articular processes. These cases are summarised in Table III. Post-mortem examination was made in the two fatal cases (Cases 5 and 6). In both, the spinal cord was severely crushed but there was no dislocation of articular processes. In Case 5 there was a large protrusion of the disc into the spinal canal. The two patients who survived had initially incomplete paraplegia, and yet there was poor recovery of function during a period of two years after injury.

Case 4. *A. G., aged* 30 *years*—Crush fracture of the sixth cervical vertebra, treated in a plaster jacket for three and a half months. He was admitted to the Spinal Injury Centre two years later with residual paralysis of both upper and lower limbs. Myelography showed a constant anterior filling defect which could hardly be explained by the residual spinal deformity, and was presumably due to disc protrusion (Figs. 4 and 5).

The evidence of this case strongly supports the view of Cramer and McGowan (1944) that cord injury in simple crush fractures is due not to pressure of the fractured vertebral body, or to dislocation with spontaneous reduction, but to compression of the ventral surface of the cord by retropulsed disc material. The treatment of compression fracture of a cervical vertebra complicated by paraplegia should therefore be on similar lines to that of compression of the spinal cord by a retropulsed intervertebral disc.

HYPEREXTENSION INJURIES IN ARTHRITIC SPINES

I have been unable to find any reference to injuries of arthritic spines except in the paper by Crooks and Birkett (1944). Nevertheless these injuries are by no means uncommon. In the present series there were six cases of this type and they were responsible for all spinal cord injuries in patients over fifty years of age (Fig. 6). All six patients had arthritic changes

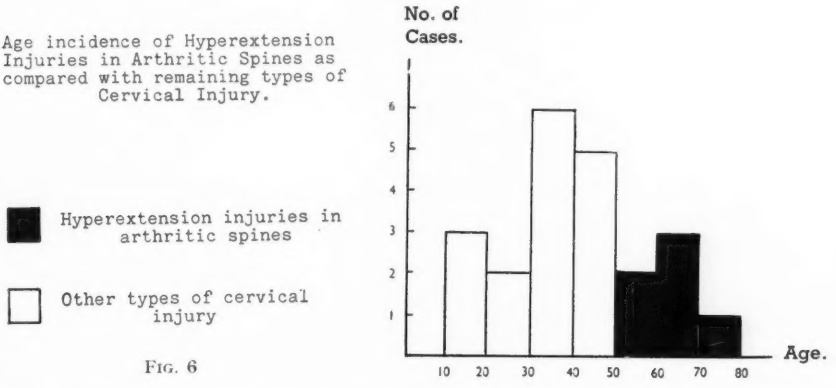


FIG. 6

in the cervical spine of moderate or severe degree, and there was strikingly little radiographic evidence of recent vertebral injury. The injury proved fatal in five of the six patients although only one had complete paraplegia. Two cases serve to illustrate the features:

Case 12. *T. B., aged* 63 *years*—Fell a distance of ten feet on to his forehead, forcibly hyperextending the neck. There was complete motor paralysis and sensory loss below the fifth cervical cord segment. Radiographs showed marked osteoarthritic changes in the cervical spine with ossification of the anterior common ligament between the fourth, fifth, and sixth vertebrae (Fig. 7). An osteophyte was avulsed from the lower anterior margin of the third cervical vertebrae; there was no other bone injury. Five hours after injury he was able to move the left leg, and tactile sensation had returned to both lower limbs. Four days after injury the patient was able to move both legs and urine was passed voluntarily. The Queckenstedt test showed no spinal block. From the eighth day his condition deteriorated steadily and he died on the eleventh day after injury.

FIG. 7

FIG. 8

Case 12. T. B., aged 63 years. Hyperextension injury of osteoarthritic spine with incomplete paraplegia. Fig. 7 shows an osteophyte avulsed from lower margin of C.3. The anterior common ligament is ossified between C.4, 5, and 6. Fig. 8 shows the autopsy specimen; the anterior common ligament and intervertebral disc between C.3 and 4 are ruptured.

Post-mortem examination revealed rupture of the anterior common ligament between the third and fourth cervical vertebrae. The rupture extended through the intervertebral disc to the posterior common ligament which was intact (Fig. 8). Abnormal hyperextension of the spine was possible at the level of injury but there was no dislocation of the articular processes. The spinal cord had a normal appearance apart from slight constriction at the level of injury.

Comment—The post-mortem findings prove beyond doubt that this was a hyperextension injury. Ossification of the anterior common ligament in the lower cervical spine accounted for localisation of violence to the upper vertebrae, and the anterior common ligament ruptured immediately above the ossified region. The cord injury was not severe, and a younger patient might have recovered.

Case 9. *T. N., aged* 58 *years*—Fell from a glass roof on to his head. There was complete motor paralysis; pain and temperature sensation were impaired below the fourth cervical cord segment; other forms of sensation were normal. Radiographs showed moderate arthritic changes in the cervical spine but no evidence of recent injury. There was no change in the neurological findings until he died eighteen hours after injury.

Post-mortem examination showed rupture of the anterior common ligament between the sixth and seventh cervical vertebrae (Fig. 9). The intervertebral disc was avulsed from the body of the upper vertebra but the posterior common ligament was intact. Abnormal hyperextension of the spine was possible at the level of injury; the upper vertebra hinged on the lower, with the articular processes acting as the fulcrum. The joint capsule of the articular processes was intact on both sides. The spinal cord had normal appearances apart from slight constriction opposite the sixth cervical vertebra; no intramedullary haemorrhage was observed on section of the cord (Fig. 10).

Comment—The post-mortem findings were very similar to those in Case 12, and proved beyond doubt that the injury was due to hyperextension violence. Neurological examination indicated that the level of spinal cord injury was three to four segments higher than that of vertebral injury, and it is therefore unlikely that the paraplegia was due to direct contusion of the cord. It is possible, however, that the cord was subjected to traction at the moment

FIG. 9

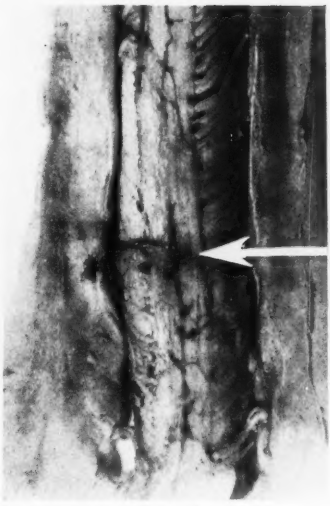


FIG. 10

Case 9. T. N., aged 58 years. Hyperextension injury with incomplete paraplegia (Fig. 9). The anterior common ligament between C.6 and 7 is ruptured and the disc is avulsed from the upper vertebra. Fig. 10 shows slight constriction of the spinal cord at the level of vertebral injury.

of extreme hyperextension, and in these circumstances the upper level of the cord lesion might well be several segments higher than the vertebral injury.

No post-mortem examination was made in the other three fatal cases but the nature of accident, and the radiographic appearances, leave little room for doubt that they too were hyperextension injuries. A summary of the clinical and radiographic features of the six cases is given in Table IV.

TABLE IV

HYPEREXTENSION INJURIES IN ARTHRITIC SPINES

Case	Age	Nature of injury	Radiographic appearances	Type of paraplegia	Result
8 J. S.	57	Fell downstairs on to face	Moderate osteoarthritis. Osteophyte avulsed from lower margin of C.6. No displacement	Incomplete	Recovery apart from residual weakness in hands
9 T. N.	58	Fell through glass roof	Moderate osteoarthritis. No fracture or dislocation	Incomplete	Death second day
10 M. C.	77	Knocked down by motor car	Advanced spondylitis. Vertebrae ankylosed below C.4. Fracture lower margin of C.3. Backward displacement of C.3 on 4 by ·5 cm.	Incomplete	Death fourth day
11 J. McQ.	67	Knocked down by car	Gross osteoarthritis. No fracture or dislocation	Complete	Death one week after injury
12 T. B.	63	Fell from ladder distance of ten feet on to head	Gross osteoarthritis. Fracture of osteophyte on lower anterior margin of C.3. No dislocation	Incomplete	Death tenth day
13 G. B.	61	Fell seven feet from scaffolding on to forehead	Gross osteoarthritis. No fracture. Widening of intervertebral space between C.4 and 5 anteriorly	Incomplete	Death second day

Mechanism of injury—Elderly patients are predisposed to hyperextension injury by two factors. With advancing years there is a tendency to kyphosis of the thoracic spine which is usually compensated by cervical lordosis (extension deformity of the neck—Fig. 12). This is a relatively fixed position, and any increase in the degree of extension is liable to cause strain or injury (Brown and Kuhns 1942). The risk of injury by hyperextension violence is greater when mobility of the spine is impaired by arthritic changes in joints and ligaments. In the event of a fall, the neck cannot be flexed in the normal defensive manner, so that when the head strikes the ground powerful angulation strain in extension is applied to the cervical region. The brunt of this violence falls on the anterior common ligament: it may rupture; or a small flake of bone may be avulsed from the lower anterior margin of the upper vertebra (Fig. 11). The tear extends backwards through the intervertebral disc, or the disc itself may be torn from the vertebra above. There is no tendency for the disc to herniate into the spinal canal as in the case of flexion injuries. Once the disc has ruptured there is little resistance to further hyperextension, and considerable separation of the vertebral bodies can occur without rupture of the lax posterior common ligament or the zygapophyseal joint capsules. Immediately the striking force is removed normal alignment of the vertebrae is restored by spasm of the neck muscles and for this reason the radiographic appearances may be deceptive. (Case 9.)

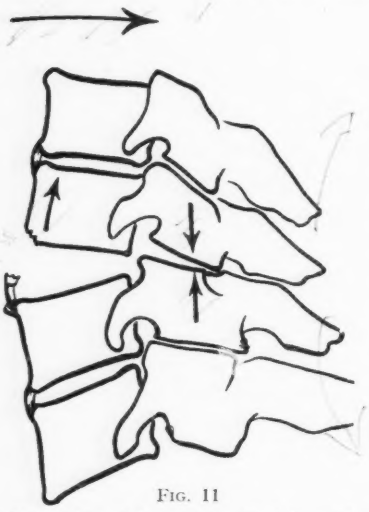


FIG. 11

Mechanism of hyperextension injury of cervical spine. The anterior common ligament is torn or avulsed with a bone fragment. There is no dislocation of articular processes.

Nature of the spinal cord injury—I have been unable to find a completely satisfactory explanation of the spinal cord lesion in these hyperextension injuries. Unlike flexion injuries there is neither gross dislocation of the vertebrae, nor retropulsion of the intervertebral disc. The cord damage is not usually severe, for the paraplegia was incomplete in five of the six patients. Two possible explanations of the spinal cord injury have been considered: 1) that it may be caused by "spinal concussion" as the result of minor displacement of a vertebral body; or 2) that it may be due to traction injury at the moment of extreme hyperextension.

FIG. 12

Tracing of radiograph of extension deformity of cervical spine compensatory to severe thoracic kyphosis. The angle between pedicles and body is more acute than in a normal spine and the spinal canal is therefore narrowed.

When there is extension deformity of the neck compensating for thoracic kyphosis the angle between the vertebral bodies and the pedicles is decreased (Fig. 12), and consequently the spinal canal is narrowed. Moreover, in hypertrophic arthritis, osteophytes may project from the posterior margins of the vertebral bodies into the spinal canal and still further reduce the margin of safety. In these circumstances it is possible that minor displacements of the vertebrae may be sufficient to concuss the spinal cord.

We have also observed that when the disc is ruptured considerable separation of vertebral bodies is possible and it is tempting to postulate that there may be traction injury of the cord. The fact that in some patients the neurological level of cord injury was several segments higher than the vertebral injury gave support to this view.

FIG. 13

FIG. 14

Steel markers have been placed in the spinal cord through the intervertebral disc between the third and fourth, and fifth and sixth vertebrae (Fig. 13). The anterior common ligament and intervertebral disc between the fourth and fifth vertebrae have been divided and the neck hyperextended (Fig. 14). There is no increase in the distance between the markers and therefore no evidence that traction force has been applied to the spinal cord.

Experiments were performed on cadavers to test the accuracy of this hypothesis. Hollow needles were inserted through the intervertebral discs immediately above and below the one which was to be divided, and were pushed on into the spinal cord. After checking the position of the needles by radiography a steel marker was introduced through the lumen of each needle into the spinal cord and the needles were removed. Further X-ray films were taken and the distance between the markers measured. The anterior common ligament and intervertebral disc between the two markers was divided. With wedges placed between the spinous processes above and below the level of section, in order to simulate the rigidity of an arthritic spine, the neck was forcibly hyperextended until there was a considerable gap between the vertebrae. Further radiographs were taken in this position and the distance between the markers again measured. Finally the spinal cord was removed and the position of the markers rechecked by radiography. These tests failed to demonstrate any separation of the markers, and the theory that paraplegia may be due to traction injury of the cord is unsupported. It must be admitted, however, that the experiments were crude, and certainly did not reproduce accurately the conditions of the living subject, so that it would be unwise to dismiss finally the possibility of mild traction lesions which stop short of neural disruption.

Treatment—There is no indication for the use of caliper traction in hyperextension injury of an arthritic cervical spine. The intervertebral disc is not displaced backwards into the spinal canal, and any vertebral displacement that there may be is corrected easily by moderate flexion of the neck. It is not necessary to use external splintage for it cannot assist restoration of cord function, and elderly patients are so intolerant of plaster jackets or collars that their application often hastens the fatal termination.

Since becoming aware of the true nature of these injuries I have been content to do no more than use a small pillow to keep the neck slightly flexed, and to arrange the usual nursing care of a paraplegic patient. It cannot be claimed that the results are any improvement on more ambitious methods, but at least it has the merit of making more comfortable the few remaining hours or days of the patient's life.

SUMMARY

Twenty-two cases of paraplegia complicating injury of the cervical column have been reviewed. The vertebral injury may be due to flexion or hyperextension violence.
Flexion injury—There are three types of flexion injury: 1) dislocation; 2) compression fracture of a vertebral body; 3) acute retropulsion of an intervertebral disc. Evidence is presented in support of the view that disc protrusion is the cause of the cord lesion when there is no radiographic evidence of bone injury, and in some cases at least when there is a compression fracture. Treatment is discussed and the indications for caliper traction and laminectomy are presented.
Hyperextension injury—There are two types of hyperextension injury: 1) dislocation; 2) injury to arthritic spines. Hyperextension injury of an arthritic spine is the usual cause of paraplegia in patients over fifty years of age. The mechanism of hyperextension injury is described. The possible causes of spinal cord injury, and its treatment, are discussed.

The author wishes to thank Mr Nichols for the clinical details of Case 1 which was under his care in the Neurosurgical Service at Killearn Hospital, and Mr J. Tulloch Brown for assistance with the experimental observations.

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PARAPLEGIA IN HYPEREXTENSION CERVICAL INJURIES WITH NORMAL RADIOGRAPHIC APPEARANCES

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This communication deals solely with a group of cases, hitherto rather obscure, in which damage to the cervical part of the spinal cord occurs without radiographic evidence of vertebral injury or displacement. Two alternative explanations of such injury have been postulated. The first is that the spinal cord pressure is due to acute massive prolapse of an intervertebral disc: but our limited experience of this lesion suggests that as a rule this *is* recognisable on radiographic examination by narrowing of the intervertebral space. The second explanation is that paraplegia is due to hyperflexion dislocation which undergoes spontaneous reduction; but it is doubtful whether this occurs at all, because forward flexion or dislocation of the cervical spine which is sufficient to damage the cord would necessarily be associated with locking of the articular facets, or crush fracture of a vertebra, or both. Neither of these lesions undergoes spontaneous reduction, and moreover both are demonstrable by radiographic examination. We suggest that the usual mechanism of these injuries, in which radiographic examination reveals no significant displacement, is forced hyperextension.

Case Report—A miner aged thirty-three years was admitted to hospital twelve hours after injury sustained by falling downstairs on his face. There was immediate and complete paraplegia to the level of the seventh cervical segment of the cord. Radiographs showed an oblique crack in the body of the second thoracic vertebra, without displacement, which obviously was not related directly to the cord damage, and a crack in the left upper articular process of the seventh cervical vertebra, also without displacement. There was no narrowing of disc spaces. Lumbar puncture showed that the cerebrospinal fluid was clear and that there was free conduction of pressure throughout the theca. Pantopaque was introduced in order to determine whether or not there was compression at the site of the cord lesion. With the patient in both prone and supine positions the oil passed freely to the foramen magnum, and it was thus clear that, whatever may have caused the original cord damage, it was no longer exerting pressure. On the third day the patient showed signs of ascending cord oedema with hyperthermia, tachypnoea, and failure of the peripheral circulation. In this state he died. Autopsy revealed that the anterior longitudinal ligament was ruptured between the sixth and seventh cervical vertebrae; the column had been torn through by detachment of the intervertebral disc from the lower surface of the sixth vertebral body. The upper segment of the column, carrying with it the intact posterior longitudinal ligament, could be displaced backwards on the lower segment with great ease, the disc remaining attached to the upper surface of the seventh vertebra and the posterior longitudinal ligament being lifted from its posterior surface (Fig. 1). At this point there was a sharply localised contusion of the cord, the principal haemorrhage being situated posteriorly, exactly opposite the sharp upper margin of the seventh cervical lamina. The displacement could be replaced easily and it tended to remain in the replaced position (Fig. 2). There is little doubt that this occurred during life, thus accounting for the normal radiographic appearances. It accounts also for the peculiarly localised cord injury which represents a sharp nip, without stretching, and is in contrast with the more diffuse contusions which are encountered in flexion injuries and in which the cord may be both stretched and compressed.

In this case the nature of the injury admits of no doubt. The mechanism is substantiated equally well in the case of a sixty-year old man who fell from a haystack and had signs of a complete transverse lesion of the cord at the fifth cervical level. Radiographs were negative, and there was no subarachnoid block. There was, however, persistent increase in the protein content of the cerebrospinal fluid obtained by lumbar puncture, and after much thought it was decided to explore by laminectomy in order to determine whether or not there was any compressing factor. Exploration was negative, and in particular

there was no intervertebral disc protrusion. Lateral radiographs of this case taken six months after injury showed a calcified shadow at the anterior margin of the fourth-fifth cervical interspace, which was interpreted as heterotopic calcification in the haematoma associated with rupture of the anterior longitudinal ligament.

In many other cases with cord damage and no vertebral displacement it has been suspected that the injuries may have been of this type, but in these instances no evidence has been available from autopsy or operation.

Discussion—It is suggested that hyperextension is not only *one* of the mechanisms of injury in the type of case under discussion, but that it is the *usual* mechanism. This has not been recognised hitherto, no doubt because two related assumptions have passed into general acceptance and have acquired for orthopaedic surgeons an almost doctrinal significance. The first is that the anterior longitudinal ligament is proof against almost any force, and that against it the extension force used in the reduction of crush fractures of the spine may be applied with safety. Thus Davis (1943) in pointing out that the anterior longitudinal ligament is incompletely elastic and has a rupture strain of 337 lb., writes: "By and large one may say that the anterior longitudinal ligament is secure against horizontal severance either of fracturing force or hyperextension reduction." Most methods of reduction, and most first-aid measures in the handling of fractured spines, are based on this belief. (Watson-Jones 1938, Taylor 1929, Platt 1938, Brookes 1937.) The second assumption is that if the cervical spine is subjected to sufficient hyperextension strain the brunt of injury will be borne by the atlas and axis vertebrae which may fracture through one or both arches or at the base of the odontoid process. Both these assumptions are partly true, but no more than partly true. The anterior longitudinal ligament is certainly strong, but it does rupture under sufficient strain, and a backward thrust applied through the head does cause dorsal

FIG. 1

Sagittal section of cervical and upper thoracic spine in hyperextension. The anterior longitudinal ligament is severed and the sixth cervical vertebra sheared from the disc below. The posterior longitudinal ligament is intact but separated from the back of the seventh cervical vertebra, thus allowing backward dislocation with narrowing of the spinal canal. Inset shows localised contusion of posterior part of spinal cord caused by momentary nipping between the posterior-inferior border of the body of C.6, and the lamina of C.7.

dislocation or fracture at the lower levels of the cervical spine. Wilson and Cochrane (1929) report, with autopsy photographs but without radiographs, a case of extension dislocation of the spine at the sixth cervical level with rupture of the anterior longitudinal ligament. W. J. Mixter (1941) envisaged such a case when he wrote, " Posterior dislocation is rare on account of the strong supporting structures, and if it does occur, I believe that spontaneous reduction might easily take place."

Diagnosis—The differential diagnosis in cases of paraplegia without radiographic evidence of significant displacement calls for reconsideration. We believe that forward dislocation due to flexion injury with spontaneous reduction is highly improbable. Damaging criticism of this conception has been advanced by Cramer and McGowan (1944). In our view the diagnosis lies between extension dislocation with immediate spontaneous reduction, and acute prolapse of an intervertebral disc. These two conditions can be differentiated with relative certainty. The history may indicate clearly whether the injury took place in flexion or extension. Severe facial injury of the type here presented suggests injury in extension. Older patients with fixed kyphotic deformity are still more liable to extension injury. Spinal manometry should be done because the results furnish a useful guide to treatment, particularly in cases of acute disc prolapse, but it does not help greatly in differentiation of the conditions under review. Disc protrusion may or may not cause spinal block, and although hyperextension dislocation does not result in continued narrowing of the spinal canal at the point of injury, yet spinal block may develop later by reason of oedema of the cord due to contusion.

FIG. 2

In the position of flexion the dislocation is completely reduced and the anatomical contours are restored. The spinal canal is no longer narrowed. There is no block on spinal manometry, and radiographs taken in this position reveal no abnormality.

Although we have stressed the normal appearances of radiographs, X-ray examination may help by disclosing minor changes. Narrowing of a disc space, or the suspicion of wedging of a vertebra, create a strong presumption that the compressing agent is a ruptured disc. The tearing of a small flake of bone from the anterior margin of a vertebral body suggests rupture of the anterior longitudinal ligament and therefore extension injury. If doubt still exists myelography should dispel it, because a prolapsed disc of sufficient size to damage the cord should be visualised without difficulty.

Mention should be made of another possibility which was suggested by Cramer and McGowan (1944), namely that " recoil " cervical cord injuries are caused by acute " piston-like " retropulsion of the intervertebral disc in hyperflexion, with immediate complete

reduction. Burns Plewes (1938) made a similar suggestion on the basis of one case studied at autopsy, but unfortunately the anterior longitudinal ligament was not examined, and it is to be noted that the patient had a wound on the forehead, thus suggesting hyperextension injury. Unless more definite evidence is forthcoming this hypothesis should be treated with some reserve. It seems most unlikely that the inelastic annulus and posterior longitudinal ligament should stretch sufficiently to allow posterior displacement of nuclear material and contusion of the cord without actual rupture.

Treatment—The practical value of the recognition of hyperextension injuries lies in treatment. The extended position in which, by well established usage, all cases of traumatic cervical paraplegia are treated is in fact the position most calculated to inflict further damage to the injured cord. If the anterior longitudinal ligament is ruptured the extended position tends to reproduce the conditions of injury, to redislocate the spine, and to narrow the spinal canal at a point where the cord normally bears the greatest ratio of size in relation to the spinal canal. We suggest, therefore, that if the diagnosis is made, or even strongly suspected, the patient should be nursed in a shell, or between sandbags, in the neutral position or even in a position of slight flexion.

The tone of this paper is perhaps dogmatic. It is based on indisputable facts, but they relate only to two cases and there is not yet ground for certainty. Nevertheless the facts have been emphasised deliberately in the hope that attention will be focused on the possibility that so-called recoil injuries may be dislocations in extension. "Recoil injury" or spontaneous reduction is clearly a reality, and it did in fact take place in our case of posterior dislocation by extension, but we remain sceptical of the occurrence of recoil or spontaneously reducing hyperflexion injuries.

Summary. 1) A case is reported of paraplegia with normal radiographic appearances in which cervical cord damage was shown at autopsy to have been due to hyperextension injury.

2) The mechanism of such injuries is discussed, together with the differential diagnosis from acute prolapse of an intervertebral disc.

3) The grave dangers of using the fully extended position of the cervical spine in the management of these cases is noted.

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PRESSURE AT THE CERVICO-BRACHIAL JUNCTION

An Operative and Anatomical Study

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To older workers the problems of the cervical rib seemed simple and straightforward. The patient complained of pain and weakness in the hand; radiographs disclosed a cervical rib, and symptoms were attributed to a hang-up of the plexus on the abnormal rib. Pressure became excessive, chiefly at the age of thirty to forty years, as the result of drooping of the shoulder girdle and it might be made worse by a post-fixed plexus. This no doubt is still true of most cases to-day, but increasing experience has shown that the subject is by no means so cut and dried as had been thought. A number of patients whose symptoms were typical showed no cervical rib on radiographic examination; radiologists reported frequent examples of cervical rib without ill consequences; and moreover it became clear that some patients were suffering from extensive thrombosis in the arteries of the hand and arm. The subject appealed to many observers and an extensive literature accumulated, much of it consisting of armchair theory supported only by isolated cases. In this article, which is based upon a fairly extensive operative and anatomical experience, an attempt is made to sort facts from fallacy in an admittedly difficult and controversial subject.

In any review it is clear that an appeal to findings at operation is of first importance. We are able to summarise the actual findings in a personal series of 122 cases operated upon by one of us (E.D.T.) between 1908 and the present date. Of these there were seventeen in which the usual symptoms were accompanied, and to a great extent overshadowed, by complications. 122 cases in 40 years

Cases with complications—Fifteen patients, all with a well-developed cervical rib, showed extensive arterial thrombosis. In twelve of the fifteen, thrombosis extended no higher than some point between the bifurcation of the brachial artery and the lower border of the pectoralis major; the second and third stages of the subclavian artery were entirely normal; no evidence of pressure was seen, and the vessel lay quite loosely on the rib. In three of the fifteen patients, gross abnormality of the artery was found—namely a thrombosed aneurismal dilatation firmly fixed to adjacent structures by much periarteritis. The upper limit of the aneurism in two of these three cases was 1 cm. distal to the rib; the artery where it lay on the rib was normal. In the third case the proximal part of the aneurism overlaid the cervical rib.

In two other patients the chief complaint was hyperhidrosis of the hand, sufficiently severe to interfere with work. In one of these, the cause was a well-formed cervical rib, and in the other a taut fibro-cartilaginous band representing the forward end of a rudimentary cervical rib.

Uncomplicated cases—These number 105 and they may be classified according to the findings at operation into several groups:

Pressure by fibrous band	12 cases
Abnormal disposition of scaleni	8 ,,
Cancellous osteoma	2 ,,
Deformed thoracic outlet, pressure of clavicle on first rib	8 ,,
No obvious cause found	5 ,,
Cervical rib	70 ,,
	105 cases

Pressure by fibrous band—In twelve patients the pressure on the lowest trunk of the plexus was due to a strong taut band springing from the tip of a small and pointed rudimentary rib. This band passed downwards and forwards in the anterior border of scalenus medius. It was easy to demonstrate that on deep inspiration the band was tautened, thus lifting the lowest trunk of the plexus which in most cases showed a permanent notch at the point of contact with the band.

Pressure due to abnormal disposition of the scaleni—In eight patients the tendinous insertion of scalenus medius into the first rib was carried forwards farther than usual with the result that the lowest trunk of the plexus and the artery were not in contact with the first rib but were raised by the narrow V formed on one side by the lateral tendinous edge of scalenus anterior and on the other by the sharp anterior border of scalenus medius. In all these cases detachment of part of both muscles resulted in cure.

Pressure due to cancellous osteoma—In two patients, both girls aged fourteen years, the cause of pressure was a large irregular cancellous outgrowth at the point where a well-developed cervical rib joined the first thoracic rib.

Pressure of clavicle on first rib in deformed thoracic outlet—In eight cases the symptoms cleared completely after resection of part of a first thoracic rib. These are probably examples of a deformed thoracic outlet, usually due to cervico-dorsal scoliosis. In six earlier operations the significance of this was not appreciated, but in the last two patients who were operated upon, distinct scoliosis with asymmetry of the thoracic outlet was recognised.

No obvious cause of pressure—In five instances most thorough exploration failed to disclose any cause of pressure and unfortunately these patients were not relieved. They may have been examples of prolapse of an intervertebral disc or brachial neuritis.

Pressure due to cervical rib—The other seventy cases showed cervical ribs in various stages of development. The word "cervical" is used in a descriptive and not in a strictly anatomical sense. It has often been pointed out that these ribs do not always spring from the seventh cervical vertebra and that occasionally they are rudimentary and deformed first thoracic ribs. This possibility can be determined easily by means of antero-posterior radiographs taken by the moving mandible technique which permits accurate counting of the vertebrae from above downwards.

In each of these seventy cases special attention was paid to the possibility of evidence of injury to the plexus or artery such as might be exerted by the clavicle. In sixty-seven cases most careful inspection failed to show outward or visible signs of injury by pressure, but the majority showed a distinct and permanent notch in the lowest trunk of the plexus where it lay against the rib. The nerve trunk had been hung up and angulated by the upper border of the cervical rib.

In three of these patients an abnormal condition of the artery was found. Beginning 1 cm. distal to the rib there was a symmetrical, fusiform aneurism extending some 2·5 cm. down the vessel; at the widest part of the aneurism the artery was dilated to fully twice its normal size. These three patients differed completely from those already described with aneurism, thrombosis, and periarteritis, in that the artery was pulsating normally and that in none was there thrombosis; the circulation was normal throughout the limb. It would seem fair to presume that these simple uncomplicated aneurisms represent an earlier stage in the pathology of thrombosis. This point may be of importance in discussing the causes of thrombosis and will be referred to later.

It is clear from this analysis of 122 operations that the causes of pressure on the neuro-vascular bundle are many and varied. No one explanation such as costo-clavicular pressure, or scalenus anterior "spasm," can account for all cases. Of the many controversial points three at least appear to be worthy of more detailed discussion, namely: 1) the scalenus anterior syndrome; 2) the costo-clavicular syndrome; 3) the cause of arterial thrombosis.

THE SCALENUS ANTERIOR SYNDROME

Although Murphy (1905) described a case in which a cervical rib was found to have compressed the neuro-vascular bundle against the lower end of scalenus anterior, and Adson and Coffey (1927) reported four similar cases, it was not until 1935 that the term "scalenus anticus syndrome" was coined by Ochsner, Gage, and De Bakey. Some confusion has since arisen in the scope and meaning of this term. There are authors who regard it as synonymous with the title of the present paper, and expressions such as "scalenus anticus syndrome with or without cervical rib" have been used. We prefer to restrict the term to those lesions in which the scalenus anterior is the sole or the main provocative factor, or in other words to those cases in which no symptoms would have arisen had it not been for this muscle.

Since 1935 much literature has accumulated, perusal of which shows so many different and conflicting views that the only conclusion left to the reader is that they cannot all be right. From this welter of opinion two groups may be distinguished. In one the action of scalenus anterior is considered to be passive and mechanical; in the other a condition of spasm is assumed. In this latter group the damage is said to be caused by active contraction of the muscle.

Passive action of scalenus anterior—Pressure on the neuro-vascular bundle by the scalenus anterior against the forward end of a cervical rib as first described by Murphy (1905) will be found occasionally in any large series of cases. It is more likely to happen if the prominence on the first rib, to which the accessory rib is articulated, is unusually large. Matters are made worse when the muscle presents a sharp tendinous edge towards the cervical rib. In an examination of sixty-four muscles we found the costal insertion of scalenus anterior to be entirely tendinous in thirty-six instances. The condition is well illustrated by the case of a girl aged fourteen years on whom we operated for very acute symptoms and found the condition shown in Fig. 1. Another factor is the variation in size of the muscle in different subjects. Increased width of insertion is very common. Reed and Weed (reported by Gage and Parnell 1947) give important figures showing the wide range in measurement; their figures agree closely with those of our own series which are:

Width of scalenus anterior insertion—0·4 to 2·5 cm.

Inter-scalene interval on the first rib—0 to 2·4 cm.

Chondro-sternal junction to medial edge of scalenus anterior—2·4 to 6·0 cm.

In view of this variability too much importance cannot be attached to reports that the scalenus anterior was found to be "hypertrophied."

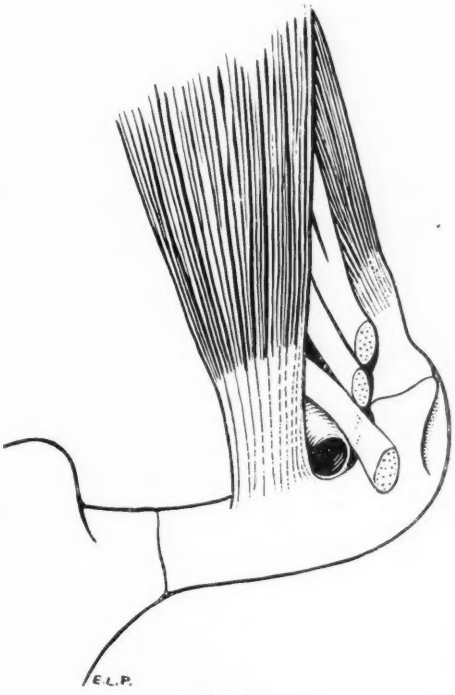

FIG. 1

Compression of the subclavian artery and inferior trunk of the plexus between scalenus anterior and the forward end of a cervical rib.

Occasionally the muscle presents a very sharp, recurved, and falciform type of insertion which might cause acute pressure on the lowest brachial trunk. This condition was noticed

twice in 102 dissections (Fig. 2). The scalenus anterior may also give rise to trouble when its insertion is wide and there co-exists an extension forwards of the insertion of scalenus medius. This is illustrated by the diagram of a dissection (Fig. 3). It results in the formation of a V on which the plexus bundle is hung up with likelihood of damage to the lowest trunk. We have met this condition at operation on eight occasions and in fifteen of 102 dissections in the cadaver. Fig. 4 demonstrates the more usual pattern of the scalene insertions.

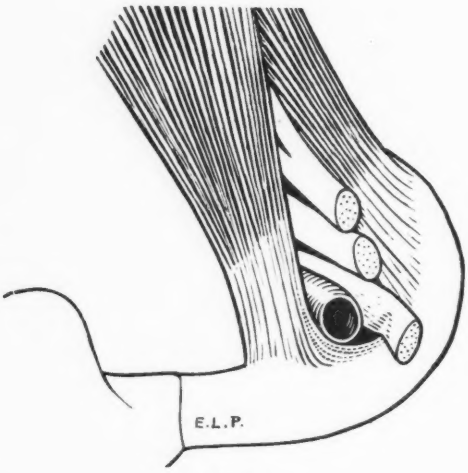

FIG. 2

The rare falciform type of insertion of scalenus anterior.

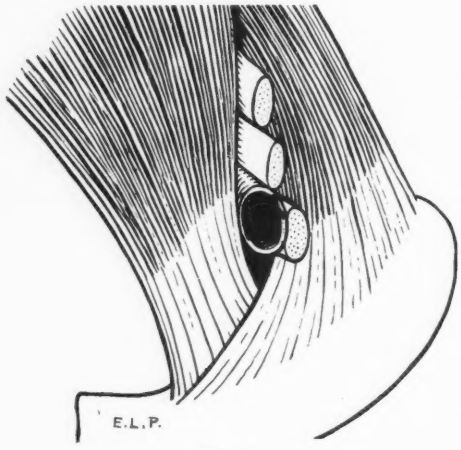

FIG. 3

The V type in which the insertions of scalenus anterior and scalenus medius overlap.

Naffziger and Grant (1938) suggest that there is a natural tendency for the neuro-vascular bundle to slide ventrally and that it is restrained by the scalenus anterior. We have never seen any shift of the bundle after cutting the muscle in the living patient, in the post-mortem body, or in the dissecting room subject.

Swank and Simeone (1944) suggested that the sixth and seventh cervical nerve roots might be compressed by the tendons of origin of the muscle. With this point in view twenty-four dissections were carried out. In twenty we found no possible evidence that nerve roots could be compressed by the tendinous origins of the muscle because they were protected by the anterior tubercles of the transverse processes. There were, however, four dissections in which some tendinous fibres were attached to the costo-transverse bar of the fifth and sixth cervical vertebrae. The tendinous slips lay between the fifth and sixth, and the sixth and seventh nerve roots respectively; they passed downwards and antero-laterally to join the lateral aspect of the rest of the muscle belly. There was no evidence of pressure on the sixth and seventh cervical nerves which were placed loosely between slips of attachment of the two scalene muscles. The disposition of these tendinous slips in relation to the nerve roots was such that it was difficult to believe that injurious pressure could be caused either at rest or in action. The impression gained was that in order to cause pressure on the nerve roots tendinous slips from the costo-transverse bars would have to be directed downward and backward to the neighbourhood of the neck of the first rib.

Very occasionally it will be found at operation that the subclavian artery appears to pass through the substance of scalenus anterior so that about one-third of the muscle lies posterior to the vessel, which is itself quite normal. In some of these the muscle deep to the

artery may be the scalenus minimus (muscle of Albinus). In 34 per cent. of our dissections we found a band of muscle arising from the transverse process of the seventh cervical vertebra and ending in a slender rounded tendon attached to a small spur on the inner margin of the first rib at a point between the two scaleni. This tendon passes between the artery and plexus. It is quite possible for this muscle to be a cause of injurious pressure. We have noted in one cadaver a groove, caused by the muscle belly, imprinted on the first thoracic nerve as it crossed the neck of the first rib (Fig. 5). Indeed Falconer and Weddell (1943) reported a case which was treated successfully by simple division of the costo-vertebral ligament the ligamentous representative of scalenus minimus.

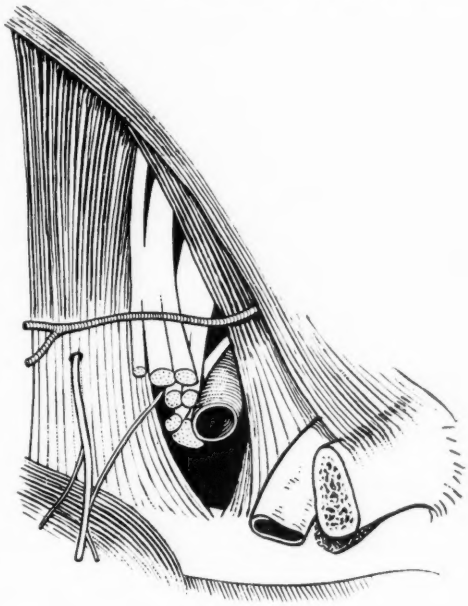

FIG. 4
The normal pattern of insertion of the scalenus anterior and scalenus medius.

From our own experience in the Neuro-vascular Clinic in Manchester we concur with Nachlas (reported by Tanna 1947) who points out that many patients have shown no improvement after scalenotomy. On the other hand there are, beyond doubt, some who have been cured. These fortunate few belong to the group in which the action of the scalenus anterior is passive and mechanical.

Active action of scalenus anterior—In this group mischief is attributed to the scalenus anterior by reason of spasm, disease, or injury. It is stated by some writers that the anterior scalene muscle holds up the first rib and that when the muscle is cut there is a notable drop of the rib. Apart from the fact that the scalenus medius which is a much more powerful muscle is still intact, we can say only that in the course of 470 anterior scalenotomies performed on the living, the great majority during thoracic sympathectomy by the anterior route, we have never seen this happen. Moreover Swank and Simeone (1944) who carried out radiographic examinations after unilateral scalenotomy say that they saw no change in the position of the rib relative to the rest of the thoracic cage. We agree entirely with their dictum: "It is unlikely that the scalenus anterior in the adult is responsible for significant independent motion of the first rib." Even if it were responsible for significant elevation of the rib it is not true

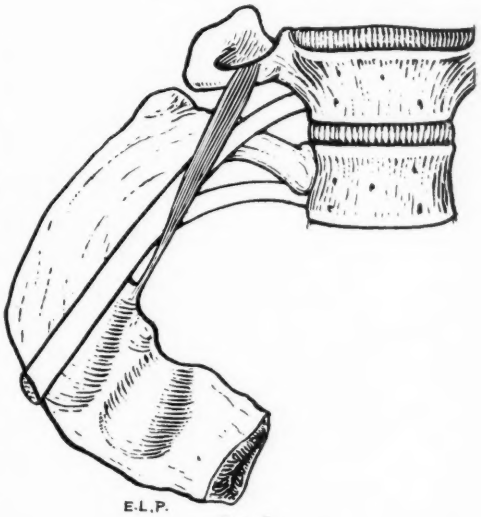

FIG. 5
Scalenus minimus (muscle of Albinus) compressing the first thoracic nerve root.

to say that when in spasm it would compress the nerve trunks against the rib. On the contrary the angle between the lateral border of the muscle and the first rib would be increased under these conditions.

If this be so, what of the theory of spasm of the muscle, and why should it develop spasm? It is not easy to understand why a relatively small muscle such as the anterior scalene should be affected by spasm while its larger companion, the scalenus medius with the same nerve supply, escapes. A vicious circle has been assumed by some observers. Brachial plexus irritation is believed to cause spasm of the scalenus anterior which in contracting aggravates irritation of the plexus. But the customary site of irritation of the plexus is far below the point of origin of the nerve supply to the muscle; and even if a source of irritation nearer to the exit of the nerves from the vertebral foramina was assumed, much more widespread spasm would be expected. In support of the theory of scalenus anterior spasm four suggestions have been made. The first is that degenerative and fibrotic changes have been found on biopsy of the muscle, but it is doubtful whether much value can be attached to these observations. Some years ago, in the course of an inquiry into the tissue changes of sclerodactyly and dermatomyositis, one of us (E.D.T.) removed for section the posterior belly of the omohyoid in eight individuals. The reports which were returned by the same experienced pathologist differed so widely that no definite conclusion could be made. A second suggestion is that there is tenderness over the muscle on deep pressure. Deep tenderness is usually present in cases of pressure on the neuro-vascular bundle, but we have found considerable difficulty in deciding that it was in the scalenus anterior and not in the trunk of the brachial plexus which is immediately adjacent. A similar objection holds for the third argument, which is that injection of the muscle with a local anaesthetic has relieved the symptoms. It is not easy to be assured that no structure other than the muscle has been anaesthetised. If radio-opaque material is added to an injection it is chastening to see on the radiographic films how widely the most precisely placed injection has spread. Fourthly it has been suggested that spasm of the muscle is caused by trauma. Not only is it difficult to envisage the cause of such injury, but it is necessary also to explain why such trauma should operate especially in women aged thirty to forty years.

In recent years there has been in Great Britain increasing doubt as to the existence of a scalenus anterior syndrome as a definite entity. From our experience we think that this attitude is well grounded. Our own view is that there cannot be justification for the pre-operative diagnosis of "scalenus syndrome" which is believed to warrant an operation directed solely to section of the anterior scalene. Such an attitude is certain to end in frequent disappointment and failure.

THE COSTO-CLAVICULAR SYNDROME

Some workers have insisted on pressure by the clavicle as an important cause of damage to the neuro-vascular bundle. Much of this writing can fairly be criticised as imaginative and unrealistic. For example, diagrams are sometimes drawn to show how the clavicle acts as one jaw of a clamp or vice compressing the artery against the first rib on depression of the shoulder. The accuracy of these diagrams is questionable because the clavicle does not travel in a two-dimensional plane; it moves in three dimensions. Furthermore, stress has been placed on the fact that in some positions of the shoulder there is diminution or arrest of the radial pulse. We hope to show that in the normal individual the clavicle is not concerned in this phenomenon which is due to causes operating distal to that bone. We have made careful and lengthy inquiry into the part played by the clavicle and our findings may be given under two headings: *a*) the effect of shoulder position on the radial pulse in normal persons; *b*) the part played by the clavicle in causing neuro-vascular complications.

Effect of shoulder position on the radial pulse—Investigations were made in 120 medical students, seventy men and fifty women, thus totalling 240 arms for examination. Oscillo-

metric observations were attempted but they were soon discarded as unreliable. In depression of the shoulder, as in carrying a heavy weight by the side, there were 116 instances of diminution and thirty-eight of arrest of the pulse; in other words 64 per cent. of examinations showed some alteration of the pulse. In retraction of the shoulder, as in " throwing a chest," the radial pulse was weakened in 134 cases and arrested in thirty; there was alteration in 68 per cent. of examinations.

The results in abduction of the limb were of special interest. In full abduction there was some alteration of the pulse in 54 per cent.; in abduction to 90 degrees the pulse was weakened in no more than 7·5 per cent. If, however, with the arm at a right angle to the body the subject was asked to adduct the arm while the observer restrained the movement, some alteration of the pulse was found in no less than 90 per cent. This would seem to indicate that the artery was compressed by muscular action. It is remarkable that arrest of the radial pulse has been tacitly assumed to be due to clavicular pressure. This cannot be true, because in cases where the radial pulse was arrested during shoulder depression it was usually easy, especially in spare and not very muscular subjects, to feel normal pulsation in the upper part of the axillary artery below the clavicle. Our attention was drawn to this fact at operation in a young man who complained of severe aching of the hand on using the arm in a dependent position. Even moderate depression of the shoulder arrested the radial pulse but left a normal pulse in the axillary artery. The upper portion of the axillary artery and adjacent nerve trunks were exposed fully and it was seen that on pulling the arm down by the side of the trunk, the artery was gripped and closed by the two heads of the median nerve.

In view of this striking and unexpected finding we decided to review the anatomy of the axillary artery. Dissections were carried out with the arm by the side. From fifty-four dissections it was found that the vessel pursued one of two different courses. In thirty-four specimens (63 per cent.) the subclavian artery, after emerging from behind the scalenus anterior, crossed the first rib obliquely and continued downwards and outwards into the axilla until it reached the neighbourhood of the second rib. Here it turned laterally for one and a half centimetres and then made a second bend to pass into the arm. In the remaining twenty dissections the vessel made its lateral curve opposite the upper part of the first interspace, near the outer border of the first rib, before continuing downwards into the axilla. This cirsoid course of the artery was described by Todd (1911) in subjects with cervical ribs and as a normal occurrence in the foetus. It appears from the present study to be a normal feature in adults when the anatomy is not distorted by abduction of the arm to 90 degrees (which of course is usual practice in the dissecting room). The cirsoid course is obviously designed to allow free movements of the shoulder in all directions without causing undue tension on the artery. Subject to individual variations the general relations of the brachial plexus to the first part of the artery in the two types were as follows. In the first the nerve cords were arranged: one postero-laterally, one posterior, and one postero-medially to the vessel. The two heads of the median nerve arose from the plexus at the point where the artery turned laterally and lay directly in front and behind the vessel, enclosing it in a scissor-like grip. Not infrequently the cords were grouped along the lateral aspect of the vessel as shown in Fig. 6, thus accentuating the gripping effect of the median nerve heads. In the second type the three nerve cords lay respectively on the anterior, postero-medial, and medial aspects of the artery causing the two heads of the median nerve to be situated antero-medial and medial to the vessel (Fig. 7).

Depression of the shoulder by pulling the arm alongside the trunk caused the axillary artery to be constricted by the heads of the median nerve in the first type. In the second, the disposition of the median nerve heads was such that constriction of the vessel could not possibly occur. Resection of the first rib caused no alteration in the plexus-artery relationship. We were not able to confirm the finding of Wright (1945) that during abduction of the

arm the axillary artery is hooked up under the coracoid process. As the arm is abducted the neuro-vascular bundle rises, but the coracoid process also moves backwards and upwards owing to scapular rotation consequent on abduction. In no dissected subject could the neuro-vascular bundle be said to approach nearer than two and a half centimetres to the coracoid process in whatever position the arm was placed.

It would appear then that approximately two-thirds of individuals possess a plexus-artery relationship such that forced depression of the shoulder interferes in some degree with the circulation through the axillary artery.

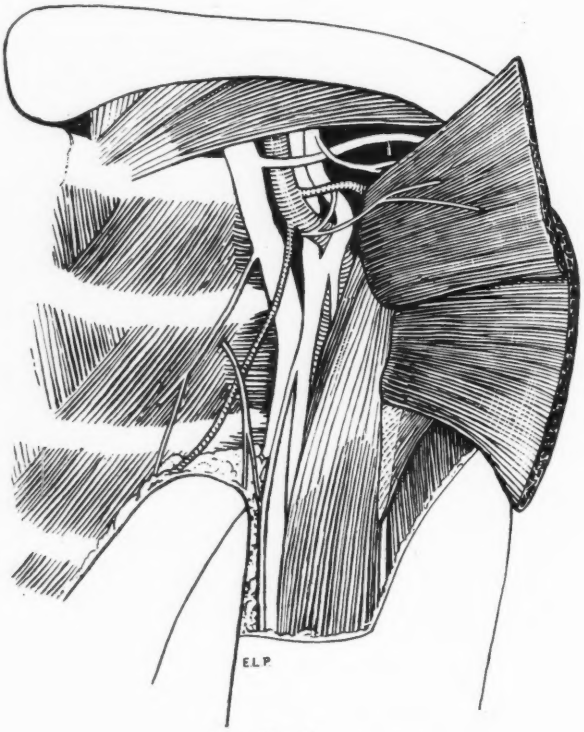

FIG. 6

Disposition of nerves relative to the axillary artery in cases where depression of the shoulder causes arrest or diminution of the radial pulse—found in thirty-four out of fifty-four dissections.

It remains to explain the alteration of the radial pulse in retraction and abduction at the shoulder. In view of the fact that excursion of the clavicle during retraction is almost identical with that during abduction it would seem likely that one explanation will hold for both movements. It is interesting to note that in many of those subjects in whom the radial pulse is modified or abolished by full abduction the alteration in pulse is temporary—that is to say that if abduction is maintained the pulse returns to normal. We interpret this temporary diminution of pulse during abduction as being due to compression between the subscapularis and pectoralis minor muscles, which are contracting as synergists during abduction, and the return of pulse to the lessening of contraction of these muscles once the position of abduction has been obtained.

This view is confirmed by the observation that in simple abduction to 90 degrees only 7·5 per cent. of subjects showed any change in the pulse, but if in this position voluntary adduction was forcibly resisted the number of instances of interference with the pulse rose to no less than 90 per cent.

Possible effect of the clavicle in compressing the artery or plexus—As a further stage of this inquiry we carried out an investigation into the movements of the clavicle in fifty bodies. In formalin-hardened bodies certain shoulder-girdle muscles had to be divided in order to allow of a full range of clavicular movement. To avoid possible error from this muscle division, ten fresh post-rigor bodies were studied in the post-mortem room.

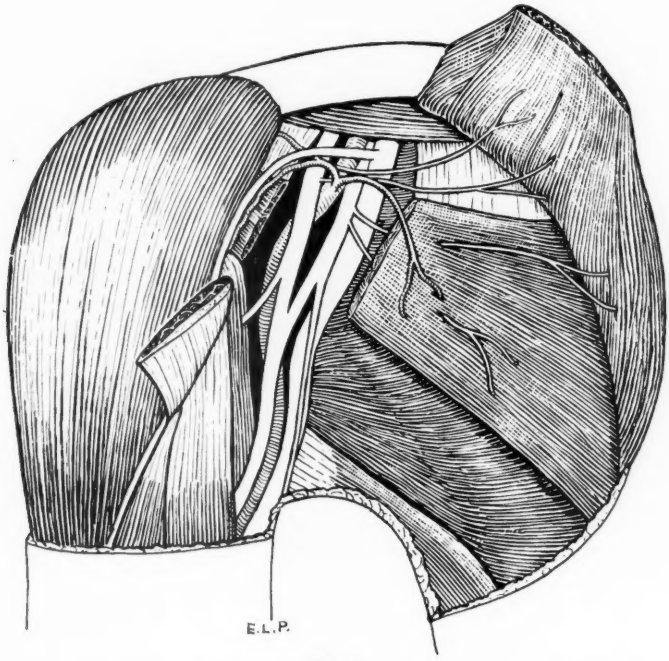

FIG. 7

Arrangement of the nerves relative to the artery which renders impossible any compression of the vessel between the two heads of the median nerve—found in twenty out of fifty-four dissections.

The findings were essentially similar in both sets of bodies. Wherever possible the measurements have been compared with similar ones obtained in the living subject, and have been found to agree very closely. Todd (1912) measured the angle of clavicular elevation by placing one limb of a pair of calipers upon the upper surface of the clavicle so as to touch the bone at the sternal and the acromial ends, and adjusting the other limb to the horizontal. He pointed out that this is an oblique measurement and does not represent the true vertical elevation of the clavicle but is the clinical measurement in the living subject.

The true vertical elevation of the clavicle can be found from radiographs or by calculation from measurements with a special instrument. We have called the angle measured by Todd's method the " clinical angle " and the elevation measured on radiographs or by instruments the " true angle " of elevation. These angles differ considerably, the " clinical angle " being the larger.

All angles and distances mentioned are average figures since individual variation is wide in the shoulder-girdle region. In the majority of adults with the shoulders at rest, true clavicular elevation, *i.e.*, the angle at which the clavicle is raised above a horizontal line passing through the upper margin of the manubrium sterni, is between 0 degrees and 10 degrees, average 6 degrees (clinical angle 20 degrees). The distance between the nearest point on the clavicle opposite to the subclavian artery, and the first rib is 3·2 cm.

When the shoulder girdle is pressed backwards in the dead body, as when the shoulders are retracted in the living, the acromial end of the clavicle rises and the bone comes to lie across the root of the neck above the level of the first rib. The true angle of clavicular elevation then measures 16 degrees (clinical angle 30 degrees) and the distance between the clavicle and first rib, opposite to the subclavian artery, is 1·7 cm. During this movement of retraction the clavicle has also rotated 5 degrees in its long axis so as to present its superior surface posteriorly. This upward and postero-medial excursion of the clavicle does not take place in a straight plane. The clavicle describes an arc pivoting on the costo-clavicular ligament.

The excursion of the clavicle during abduction is essentially similar to that during retraction. The true angle of elevation, however, is increased to 22 degrees (clinical angle 40 degrees) and rotation in the long axis of the bone measures 30 degrees, owing to the extent of scapular rotation on the chest wall consequent on full elevation of the arm. With regard to the suggestion that the clavicle compresses the artery against the first rib during these movements the minimum distance measured between the two bones was 1·5 cm. At no point did the clavicle impinge on the subclavian artery.

When considering the question of costo-clavicular pressure, clavicular rotation is an important event. With the shoulders at rest the medial two-thirds of the bone is curved with its convexity directed anteriorly. When the clavicle lies across the root of the neck this convexity forms an arch over the neuro-vascular bundle, owing to the rotation that has accompanied full abduction or retraction of the shoulder (Fig. 8). Doubtless this is nature's method of avoiding pressure by the bone structures on the soft parts.

During the movements of abduction and retraction of the shoulder it was noticed that the tendon of the subclavius muscle compressed the subclavian vein against the first rib (Fig. 6) but at no point did the clavicle itself interfere with this vessel.

From the position of full abduction or retraction to that of full depression of the shoulder, the clavicle describes an arc moving downwards and forwards. The more the shoulder is depressed the more forward does the clavicle move, thus widening the costo-clavicular interval. At full depression this measured 3·7 cm. opposite the subclavian artery, *i.e.*, an increase of 0·5 cm. when compared with the position at rest. In order to press the clavicle directly on to the vessels and nerves as they cross the first rib it was necessary to open the sterno-clavicular joint and sever the intra-articular disc.

With regard to the plexus and artery during depression of the shoulder it was seen that the superior and middle trunks, together with the contribution from the seventh cervical root to the nerve to serratus anterior, were tightly stretched across the tendinous antero-medial edge of the scalenus medius (Fig. 4). The inferior trunk was pulled down into the angle of junction between the scalenus medius and the first rib. It was not possible by this manoeuvre to compress the artery against either of the scalene muscles; nor was there any evidence that the plexus moved anteriorly toward the costal insertion of the scalenus anterior when the arm was depressed. Thus it will be appreciated that when the shoulders are retracted or abducted the girdle moves *upwards* and postero-medially, and during depression in the reverse direction. Such statements which have appeared in the literature on this subject as " downward and backward retraction " or " downward and backward bracing " of the shoulders can therefore have no meaning.

It would seem that in individuals with normal anatomy the clavicle can play no part in compressing the artery or plexus against the first rib. Only if the costo-clavicular interval is narrowed, from any cause whatsoever, is it possible that the movements of retraction and abduction of the shoulder may lead to pressure symptoms.

Fresh interest has recently been aroused in movements of the clavicle in relation to the first rib by reason of the large number of patients with paraesthesia of the hands who have

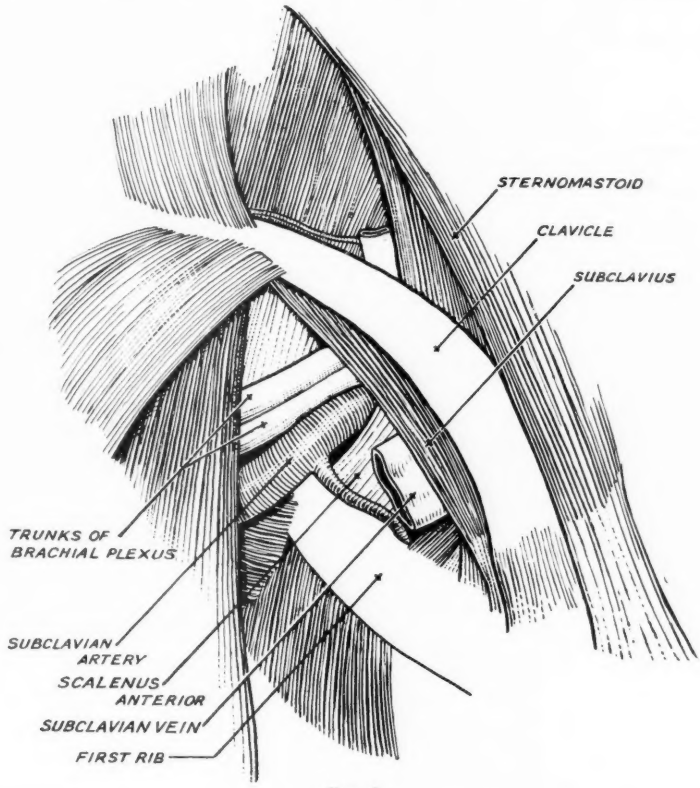

FIG. 8

Illustrating how rotation of the clavicle in its long axis causes the anterior convexity of its medial two-thirds to form an arch over the neuro-vascular bundle (arm abducted to 90 degrees).

been seen in neurological and neuro-vascular clinics in Great Britain during the latter part of the war and since that time. They are almost entirely middle-aged women suffering from overwork and under-nourishment. They have been forced to do unaccustomed heavy work, to stand for many hours in queues, and to carry heavy shopping baskets. The pain is severe and it often precludes proper sleep. Many have discovered trick positions of the arms which give some ease, the one feature common to these positions being moderate but seldom complete abduction, thus taking strain off the shoulder-girdle muscles. The pain in these cases is not due to clavicular pressure because in a normal individual the more the shoulder is depressed the wider becomes the costo-clavicular interval. They are, we believe, due simply to drag of the plexus across the tendinous anterior edge of the scalenus medius such as is seen when the arm of a dissected body is pulled downward. Loss of tone in the shoulder-girdle musculature in these middle-aged women is no doubt an important contributory factor.

THE PROBLEM OF THROMBOSIS

Arterial thrombosis is an occasional and grave complication of cervical rib. In our 122 patients it was found in fifteen (12 per cent.). Its explanation is not yet fully understood. Todd (1913) believed that paralysis of sympathetic fibres led to thrombosis. Telford and Stopford (1931) carried this work a stage further and pointed out that a leash of undistributed sympathetic fibres was sometimes found in the lowest trunk of the brachial plexus at the point of pressure. Irritation of these fibres would lead to arterial spasm and finally thrombosis, as occurs in ergot poisoning and in old-standing Raynaud's disease. This view was confirmed by Blair, Davies, and McKissock (1935) who, however, thought that the pressure resulted in neuritis of that part of the trunk which contained the sympathetic fibres.

This idea of sympathetic irritation was strongly criticised by Lewis (1934) and later writers, who held that the cause of thrombosis was serious direct injury to the artery where it crossed the rib. Certain facts may, however, be accepted as evidence in favour of the sympathetic irritation theory. We have observed very gradual onset with pallor and coldness of the hand, increasing at the end of a day of hard work, and showing much recovery after the night's rest. The variability is such that by some writers the condition has been included, we think wrongly, as one form of the Raynaud phenomenon. Moreover in twelve of our fifteen operations for thrombosis, not the slightest evidence of damage to the artery where it crossed the rib has been seen, but all cases have shown a large cervical rib actually grooved by the lowest trunk of the plexus (Fig. 9). Furthermore the occurrence of two cases of hyperhidrosis, cured by removal of pressure on the lowest trunk of the plexus, is strong evidence for the theory of sympathetic irritation. There would seem to be good grounds for the view that in some cases, probably the majority, the cause of thrombosis is long-continued intermittent arterial spasm.

FIG. 9

Excised portion of a cervical rib showing the groove made by the inferior trunk of the plexus.

This view, however, will not explain the six cases in our series of operations in which gross abnormality was found in the artery. It will be remembered that of these, three were simple aneurismal dilatations without vascular complications, while the other three showed thrombosis with much periarteritis and extensive thrombosis lower down the arm which was no doubt embolic in origin.

It seems reasonable to suppose that the simple aneurism, uncomplicated by thrombosis, represents an earlier stage in the evolution of the disease. No doubt, after a time, the aneurism becomes thrombosed suddenly as the result of further stretching or repeated trauma. The final incident is rupture of the intima with immediate thrombosis. In this connection a recent experience is of interest. A girl aged fourteen years had for some months devoted all her spare time to playing a primitive game of fives against a wall of the house with a soft rubber ball. Her mother had reproved her frequently for wasting so much time. One day she played both morning and afternoon, and in the evening came into the house saying that her left hand was numb and cold. Her mother noticed that the hand was white but she did not seek advice for forty-eight hours, by which time there had appeared two black patches, one on the pulp of the index finger and a second on the dorsum of the hand. She was admitted to the Neurovascular Clinic of the Manchester Royal Infirmary as an emergency and operated upon at once. Springing from the seventh cervical vertebra there was a large true cervical rib which articulated at its anterior extremity with a prominent boss on the first thoracic rib, just lateral

to the scalene tubercle. The segment of the artery lying on the rib was normal but just below the rib there was an aneurismal dilatation solidly thrombosed and with much recent acute periarteritis (Fig. 10).

The anterior end of the cervical rib was removed together with the prominence on the first rib. The gangrenous patches resolved quickly and within a month the limb, although without palpable pulse, was normal in appearance and function. The girl was asked to show how she played the game; it was clear that each blow given by the hand to the ball was preceded by strong retraction of the shoulder by which to reinforce the stroke, thus leading to trauma between the clavicle and the cervical rib.

The aneurism found in this case must have existed for a long time and caused no symptoms until the final and sudden catastrophe of thrombosis. It is likely that sudden and massive clotting followed rupture of the overstretched intima.

It is interesting to speculate as to why an aneurism of the vessel may be formed distal to the point of contusion. A link is at once suggested with the experiments of Halsted (1916). He demonstrated that after constricting an artery by an encircling metal band the vessel showed dilatation distal to the band. Halsted explained this result by physical changes in the blood-flow. It may not, however, be safe to apply deductions from the experiment of a permanent metal band to cases where the cause is an infinite series of small contusions of the arterial wall.

It has been suggested that the aneurism may be caused by weakness consequent on damage to the vasa vasorum. In uncomplicated aneurisms, however, there is no naked-eye evidence to support this theory; the arterial wall appears uninjured; it is resilient and shows no evidence of ill-health. We would suggest that the cause may lie in paralysis of the vaso-motor nerves which degenerate as the result of repeated contusions. Secondary dilatation and stretching of the denervated middle coat would extend distal to the site of injury just as far as the point at which the next nerve relay came in. Kramer and Todd (1914) found that the nerve supply of the subclavian artery and proximal part of the axillary artery was derived from the inferior cervical ganglion by a perivascular plexus, and that the first recruitment from a somatic nerve entered the distal portion of the axillary artery. As would be expected considerable variation was found in the origin and distribution of the nerve filaments running to the vessels. We have repeated the work of Kramer and Todd with respect to the subclavian, axillary, and upper portion of the brachial arteries, checking the findings in our dissections by histological examination of such fibres as we took to be nerves. The importance of this is shown by the fact that quite frequently a filament, confidently assumed to be a nerve, proved on section to be a minute artery—a vas nervorum surrounded by its fibrous sheath.

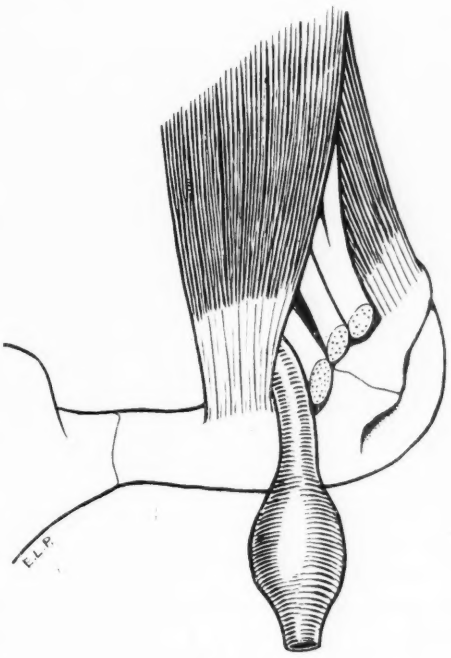

FIG. 10

A typical aneurism. Drawn from the case of the girl described in the text.

We found, as did Kramer and Todd, that dissection of the formalin-hardened cadaver was profitless for this type of work. Accordingly fresh material was obtained from eleven post-mortem bodies. We confirmed the fact that the subclavian artery and the proximal part of the axillary artery are supplied by a perivascular plexus derived from the inferior cervical sympathetic ganglion. From four of the post-mortem specimens filaments running from the plexus to the axillary artery were dissected and proved on section to be definite bundles of nerve fibres. The first of these was derived from the lateral cord of the brachial plexus and entered the arterial coat at a distance of 3 cm. from the outer border of the first rib. The second was given off from the lateral pectoral nerve and entered the arterial coat 3·5 cm. from the outer border of the first rib. The third, arising from the loop of communication between the lateral and medial pectoral nerves, entered the vessel wall 3 cm. from the outer border of the first rib. The fourth, which arose in a similar fashion to the third, was traced for a distance of 7·5 cm. along the vessel before it entered the arterial coat. These findings seem to show that reinforcements of sympathetic fibres from somatic nerves reach the artery sufficiently high to limit the area liable to aneurismal dilatation, to a comparatively short distance beyond the point of trauma.

CONCLUSIONS

While the diagnosis of a pressure lesion at the cervico-brachial junction is usually simple, the causes of such pressure are so varied and obscure that in the present state of our knowledge it is not possible to forecast the exact cause in all cases. It can be said only that symptoms which are predominantly in the hand point to a hang-up of the lowest trunk of the brachial plexus on some abnormal structure, while more widespread symptoms such as pain in the region of the upper arm indicate clavicular pressure during retraction and abduction of the shoulder on an abnormal structure, most commonly a cervical rib.

Having decided on clinical grounds, radiographic evidence, and the failure of conservative treatment, that operation is indicated, the approach should be frankly exploratory through an adequate incision. To operate with no more purpose than to carry out a preconceived plan such as anterior scalenotomy is to invite failure.

SUMMARY

1. The causes of pressure on the neuro-vascular bundle of the upper limb are many and varied. No one cause such as clavicular pressure can explain all cases.

2. Costo-clavicular pressure is not possible when there is a normal first rib and a normal thoracic outlet, but it is certainly a factor when the costo-clavicular interval is narrowed by the presence of a large cervical rib or an abnormal first thoracic rib. Clavicular pressure can act only during retraction and abduction, not in depression of the shoulder.

3. Temporary alterations in the radial pulse on movements of the shoulder in normal individuals are due to causes distal to the clavicle and have no relation to costo-clavicular pressure.

4. While irritation of sympathetic nerve fibres may explain the majority of cases of thrombosis, there are others in which clotting occurs in an aneurismal dilatation produced by pressure between the clavicle and the abnormal costal element. It is likely that the thrombosis occurs in an aneurism which has been present for some length of time. The cause of the aneurismal dilatation may be vaso-motor paralysis of a segment of the artery, ending distally at a point where a fresh intact leash of nerves is relayed to the vessel.

5. The importance of the scalenus anterior syndrome has been over-emphasised. If operative treatment is limited in all cases to anterior scalenotomy the results will be disappointing.

6. If operation is advised it should be performed without rigid and preconceived ideas, through an adequate incision, and with exploration wide enough to allow thorough investigation of the cause of pressure.

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FATIGUE FRACTURES OF THE FIBULA

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In describing certain fractures of the fibula, sustained without violence, the writer (Burrows 1940) expressed the hope that further cases would be published so that something could be learnt of these uncommon fractures. Additional cases have now been described. The writer has encountered five other personal cases, and scrutiny of the literature has revealed at least one previous case (Detlefsen 1937). It seems fitting, therefore, to review the subject in detail. Fatigue fracture * of this bone ranks in frequency after the metatarsus, the tibia, and perhaps the femur (Asal 1937, Richmond 1945). As a site for fatigue fracture the fibula has a special, though not unique, place in being a non-weight-bearing bone—a fact which emphasizes the importance of the stresses of muscular action rather than those of weight-bearing.

FATIGUE FRACTURE OF THE LOWEST THIRD OF THE FIBULA

Detlefsen (1937) described an overloading fracture 6 cm. above the lower end of the fibula which developed without injury in a compulsory state worker. He compared it with the fatigue fractures of inanimate materials in industry.

In 1940 Weaver and Francisco described the case of a student, aged twenty-two years, who had been carrying out running practice for several months when for no apparent reason he sustained what was almost certainly a fracture of the right fibula, 3 cm. above its lower end. Five months later a similar condition developed in the neck of the left fibula. The authors regarded the lesions as " pseudo-fractures " and ascribed them to non-suppurative osteomyelitis.

Simultaneously and independently the present writer (Burrows 1940) described two cases of spontaneous fracture of the apparently normal fibula in its lowest third, developing with no history of injury but in association with much activity (cross-country running in one case). While recognising that two cases alone warranted speculation rather than conclusion he suggested an analogy with march fracture of a metatarsal bone. He compared the condition with other military fractures occurring without obvious violence, and indicated the desirability of recording further cases.

Detlefsen (1941) described fatigue fracture of the lowest third of the left fibula in a woman thirty-seven years of age whose work at a machine called for depression of a vibrating pedal with the left foot for eight hours daily. Ingersoll (1943) described fatigue fractures in the lowest two or three inches of the fibula of three patients—all boys, all aged nine years, and all beginners at ice-skating. In one case the fracture was bilateral.

In 1944 Hamilton and Finklestein described bilateral fatigue fracture of the lowest third of the fibula in a recently recruited infantryman who was undergoing training and subsequently suffered march fracture of the second metatarsal bone. The fibular fractures were described as march fractures. In another bilateral case recorded by Ronald (1945) the symptoms began during a cross-country run and changes developed in the right fibula about two weeks after those in the left.

Richmond (1945) described two unilateral cases in soldiers undergoing training. In one, the pain developed after a cross-country run. McPhee and Franklin (1946) recorded cases in a track runner and a basketball player.

* *Spontaneous fractures* are fractures without material violence (*i.e.*, without acute trauma), and may occur in pathological bone or in seemingly normal bone. *Fatigue fractures* are spontaneous fractures of seemingly normal bone. They appear to result from a summation of stresses, which singly are insufficient to produce fracture.

TABLE I—Analysis of Published and Personal Cases of Fatigue Fracture of the Fibula
(*Doubtful cases in italics*)

Part of fibula affected	Author	Year	No. of pts.	Side	Age (Yrs.)	Sex	Occupation	Activities
	Grunert	*1910*	*1*	*R*	—	*M*	*Grenadier recruit*	*Doubling*
	Finny	*1924*	*1*	*R and L*	—	*M*	*Infantry private*	*Cross-country running*
	Detlefsen	1937	1	—	20	—	Compulsory state service	
	Weaver and Francisco	1940	1	R	22	M	Student	Running practice for "Track team"
	Burrows	1940	2	L R	61 17	F M	Parish worker Schoolboy	Much walking Cross-country running
	Detlefsen	1941	1	L	37	F	Muster needlewoman	Depression of vibrating pedal
(i) Lowest third	Ingersoll	1943	3	R and L R L	9 9 9	M M M	Schoolboy Schoolboy Schoolboy	Beginner at ice skating Beginner at ice skating Beginner at ice skating
	Hamilton and Finklestein	1944	1	R and L	—	M	Infantry recruit	Training
	Richmond	1945	2	L L	— —	M M	Infantry N.C.O. training Infantry soldier training	Cross-country running Assault course, etc.
	Ronald	1945	1	L and R	—	M	(Not stated? airman)	Cross-country running
	McPhee and Franklin	1946	2	R R	— —	M M	University athlete University athlete	Track running Basketball
	Burrows	1948	5	R R L R R	20 45 56 51 63	M F F F F	Medical student Nurse Housewife Housewife Housewife	Cross-country running Much on feet Household duties and ,, ,, walking Household duties
	Hopfengärtner	1907	39	—	—	M	Infantry in first year	Jumping (25 cases)
	Dreist	1909	1	—	—	M	Fusilier in first year	Attacking from kneeling
	Grunert	1910	1	R	—	M	Grenadier recruit	Training
	Scherf	1933	9	4R 5L	— —	M M	Artillery recruits	Jumping exercises from double-knees-bend
	Asal	1936	"Many"	2 bilateral	—	M	Artillerymen (mostly)	Jumping on and off limber for hours on end
(ii) Uppermost third	Asal	1937	12	"Many" bilateral	—	M	Artillerymen (mostly)	Gun exercises, with much jumping on and off limber
	Wachsmuth	1937b	1	L and R	21	M	Infantry recruit	Training exercises
	Wachsmuth	1937a	"Several"	—	—	M	Infantry in training and artillerymen	Jumping, knee-bending, squatting
	Detlefsen	1937	1	R	21	—	Compulsory state service	
	Weaver and Francisco	1940	1	L	22	M	Student	Running practice for "track team"
	McPhee and Franklin	1946	1	L	—	M	University athlete	Running
	Siemens	1942	1	L and R	16/12	M	(Infant)	Reputedly very lively
(iii) Middle third	Leveton	1946	1	L	19	M	Recruit	Training
	McPhee and Franklin	1946	1	L	—	M	University athlete	Running

FIG. 1

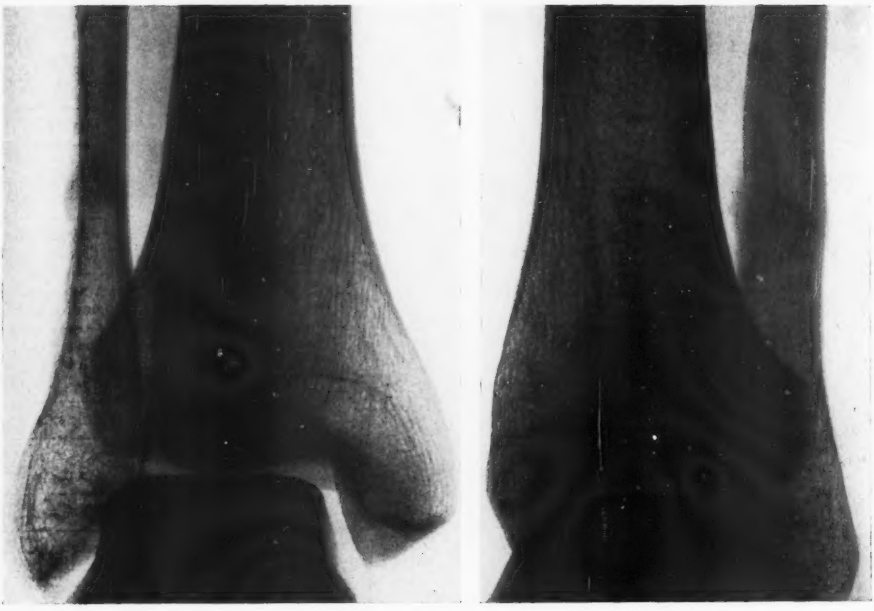

FIG. 2

Case 1. Fig. 1 shows the radiographic appearances twenty-three days after the first symptoms and sixteen days after the first acute symptoms. A band of slightly increased density and subperiosteal ossification is just discernible. A week before, no radiographic abnormality could be detected. Fig. 2 shows the appearances five weeks later than those in Fig. 1. Calcified callus is much increased but no fracture line is evident.

Association with strenuous running in at least five cases may throw new light on two examples of fibular fracture which were reported many years ago. Grunert (1910) reported a fracture at the junction of the lowest and middle thirds of the fibula in a recruit who complained of pain after running, but he ascribed it to forced supination. Finny (1924) told of a soldier who was running in an inter-regimental cross-country race and twisted his left foot inwards on a lump of earth; as he stumbled the other foot was twisted in a similar manner. He completed the remaining three miles of the race and finished twelfth out of a large field. Radiographs showed a fracture two inches above the lower end of each fibula.

Further Personal Cases

Five additional cases of fatigue fracture of the fibula at this site are here reported.

Case 1. Medical student, aged 20 years. Nine years before being seen he won a seven-mile inter-club cross-country race in 40 mins. 44 secs. As he jogged back to the pavilion, which was about a mile distant, the lower end of the right fibula became painful and the ankle felt stiff. After being supported with elastic adhesive strapping it improved each day and four days later, with the ankle still strapped, a gentle three-mile run caused no discomfort. Two days before being seen he had taken part in an inter-university cross-country run of more than six miles. After running three and a half miles sudden pain at the same site made him stumble but not fall. There was no audible snap. He had to walk home, covering three miles of frozen rough ground in about an hour. The strapping became tight and the painful area throbbed. Change of strapping and a hot bath brought relief, but he was awakened at about 2 a.m. with much pain which was relieved by removal of the strapping. There was no further improvement. Pain increased on resuming activity after rest, or moving the limb forwards against gravity. There had been no injury and the pain had preceded the stumble. *On examination* the fibula was tender over a length of about one and a half inches, centred at a point of maximum tenderness two and a half inches above the tip of the lateral malleolus. No swelling was visible or palpable and there was no redness or increased skin temperature. Active and passive tarsal and ankle movements were full except that active dorsiflexion was limited at the right angle; passive dorsiflexion alone gave slight pain at the site of tenderness. Similar pain was produced by jarring or by "springing" the tibia and fibula. *Radiographs* showed no bone abnormality even on close scrutiny with a lens, but the *clinical diagnosis* of fatigue fracture of the lowest third of the fibula was made with confidence. Elastic adhesive strapping was applied from the metatarsal heads to the top of the calf with steady improvement in the pain. Seven days later there was still no radiographic change; but seven days later, when a slight hard swelling had appeared at the tender site, *further radiographs* (Fig. 1) showed a band of slightly increased density about three-sixteenths inch broad, with very slight subperiosteal ossification situated two and a half inches above the tip of the lateral malleolus. By the end of the next three weeks the range of movement had returned fully, the radiographic changes were substantially increased, and the strapping was discarded. After yet another fortnight, walking, standing and running were painless though followed by throbbing; movements were full; and radiographs showed increased subperiosteal opacity but still no fracture line (Fig. 2). After another fortnight there were no symptoms or signs except slight local tenderness and bony swelling. These were still evident fifteen weeks after the onset when radiographs showed that the new bone was well organised and with some indication of a transverse, relatively clear, band. Twenty-two weeks from the onset he was free of all symptoms and won the inter-hospitals three miles race in 16 mins. 3¼ secs. A bone ridge was still palpable clinically and visible radiographically. At no time was wasting of the calf demonstrable. *Control radiographs* of the hand showed no evidence of porosis.

Case 2. Nurse, aged 45 years, working in an old-fashioned nursing home, who had not recently increased her activities or suffered any injury. One afternoon, four weeks before examination, pain and swelling developed suddenly in the region of the right lateral malleolus. Both were worse by day than by night. She managed her duties for two weeks, and the swelling diminished gradually. *On examination,* oedema and a slightly raised cutaneous temperature were found over the lower part of the right fibula which was tender at a level about one and a quarter inches above its lower end. Ankle and tarsal movements were full and painless. *Radiographs* (Fig. 3) showed a fracture of the fibula one and a half inches above the tip of the lateral malleolus. Displacement was minimal. In the antero-posterior view the fracture appeared transverse but the lateral view showed that it passed obliquely forwards and downwards for a quarter to half an inch. Scarcely any callus was visible. Strapping, which had been applied to the foot, ankle, and calf, was removed by the patient within a week because she had become quite comfortable. When seen again three and a half weeks after the first occasion, and seven and a half weeks

from the onset, she was quite comfortable and the oedema had disappeared; but there was still slight tenderness and by then some bony swelling at the site of fracture. *Further radiographs* showed a fairly pronounced cuff of callus (Fig. 4).

FIG. 3

FIG. 4

Case 2. Fig. 3 shows the radiographic appearances at first examination, four weeks after the onset of symptoms. A small amount of ossified callus is present. Fig. 4 shows the appearances three and a half weeks later than those in Fig. 3 and seven and a half weeks after the onset of symptoms. Ossified callus has increased considerably.

Case 3. Housewife, aged 56 years, who was "on the go all the time" and "did everything" for a husband and four bachelor sons. She liked walking, and five weeks before being seen she took an unnecessary walk of two miles in addition to ordinary activities. On rising next morning she had great difficulty in carrying on because of pain in the left ankle which soon became swollen, shiny, and hot, but not reddened. The pain was relieved by rest but it was particularly severe when activity was first resumed. Walking was worse than standing. There had been no injury. Slight spontaneous improvement occurred slowly. The diagnoses of gout and thrombophlebitis had been made at different times. *On examination,* five weeks after the onset, diffuse pitting oedema of the left ankle region was found with tenderness just proximal to the lateral malleolus, and with about ten degrees limitation of plantar flexion movement. The feet were supple but were flat both longitudinally and transversely. *Radiographs* showed a broad transverse fracture line, one and a half inches above the tip of the lateral malleolus, without displacement and with little callus (Fig. 5). *Control radiographs* of the hand showed no porosis. Elastic adhesive strapping was applied from the metatarsal heads to the upper calf for three weeks with improvement. The pain did not disappear completely, nor did plantar flexion return fully for twenty-five weeks. Even then very slight local tenderness and bony swelling persisted. *Further radiographs* (Fig. 6) taken nine months after the onset showed complete restoration of bone texture, the only indications of the old fracture site being a band of slightly increased density and very slight bony thickening.

Case 4. Housewife, aged 51 years—a very energetic woman. Besides caring for a husband and two children aged twenty-one years and thirteen years, she worked five hours a day as inspector in an engineering works—a sitting job which involved a brisk twenty minutes' walk each way over a rough country road. On getting out of bed thirteen days before being seen she found that the lower right fibular region was painful. She went to work but increasing pain during the afternoon drove her to her doctor who diagnosed "sepsis." She remained off work (but continued housework) and applied kaolin poultices for a week, and then a crêpe bandage, without improvement. Walking was more painful than standing, and rest from weight-bearing brought improvement. Nocturnal aching did not interfere with sleep. The pain was not aggravated on first resuming activity after rest. There had been no relevant injury. *On examination*

FIG. 5

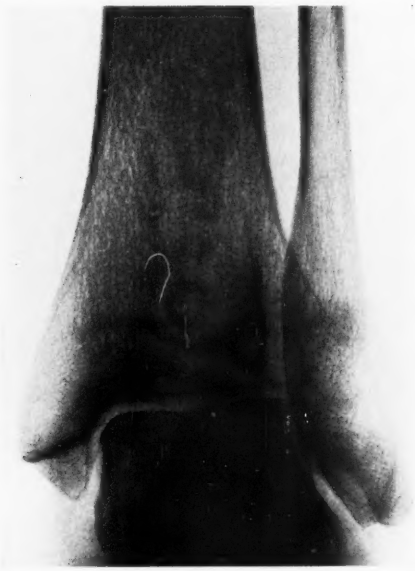

FIG. 6

Case 3. Fig. 5 shows the radiographic appearances five weeks after the onset. Though the fracture zone is distinct, callus is scanty. Fig. 6 shows the radiographic appearances nine months after the onset. The structure of the bone is fully restored but the site of fracture is still marked by a dense band.

FIG. 7

Case 4. Radiographic appearances twenty-five days after first examination. Horizontal fracture one and a half inches above the tip of the lateral malleolus with substantial callus.

non-pitting oedema was found over the outer part of the right ankle region without redness or increased cutaneous temperature. Localised tenderness was elicited about one and a half inches above the tip of the lateral malleolus. Dorsiflexion and plantar flexion were about half the normal range, inversion and eversion about three-quarters. Moderate hallux valgo-rigidus was present. *Radiographs* showed an almost horizontal fracture, one and a half inches above the tip of the lateral malleolus with slight posterior displacement of the distal fragment. *Control radiographs* of the hand revealed no porosis. A plaster-of-Paris walking cast was worn for eleven days, followed by elastic strapping. After twenty-five days' treatment there were still some pain, local tenderness, and swelling. The girth of the calf was diminished by a quarter of an inch. Movements were full and painless except that inversion caused pain at the fracture site. *Further radiographs* (Fig. 7) showed substantial callus. Thereafter she had practically no discomfort, and returned to work nine days later. After another five days there was still a hard, slightly tender swelling on the fibula and still a quarter of an inch wasting of the calf, but movements were full and painless.

FIG. 8

Case 5. Radiographic appearances.

Case 5. Housewife, aged 63 years, with only a husband to look after but nevertheless on her feet almost all day. Twenty-four days before being seen she was walking about on household duties when she noticed severe pain in the outer side of the lower part of the right fibula. The pain, which drew attention to swelling, was aggravated by standing or walking, especially out-of-doors, and was improved by rest. There had been no injury. *On examination* the right fibula presented a bony hard, tender swelling, one and a half inches from its lower end, with adjacent non-pitting oedema but with no redness or raised temperature. There was a full range of painless tarsal and ankle movement and no measurable wasting of the calf. *Radiographs* (Fig. 8) showed an almost transverse fracture about one and a half inches above the tip of the lateral malleolus without displacement. A little calcified callus was present. There also appeared to be diminished density of all bones, the same being so on the left side but without fracture. *Control radiographs* of one hand (Fig. 9) showed evidence of slight generalised porosis which, however,

was not considered to be of pathological degree. Elastic adhesive strapping was applied from the base of the toes to just below the knee. It was continued for five weeks by which time all pain, tenderness and oedema had subsided. The patient reported that twenty years earlier swelling of one foot had appeared spontaneously and without injury. It had lasted about one month and no radiographs had been taken. Radiographs now show evidence of a united fracture of the distal third of the second left metatarsal bone (Fig. 10).

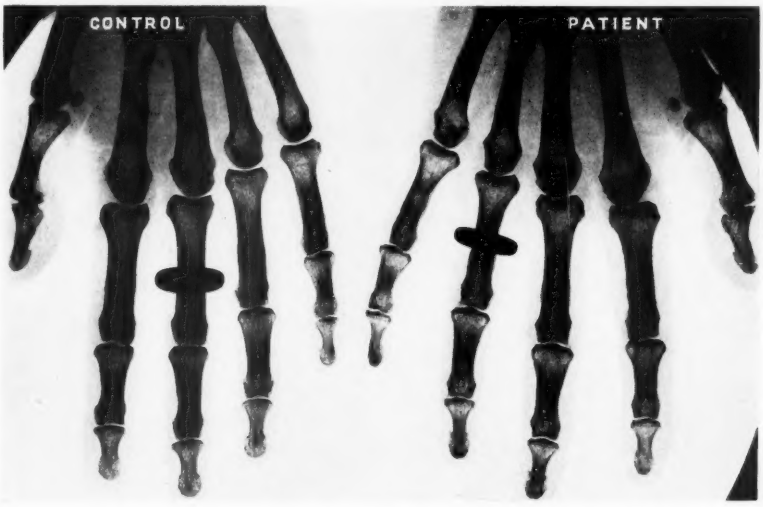

FIG. 9

Case 5. Control radiograph of hand. The patient's hand (which was taken simultaneously with that of a control subject on the same film) shows very slight relative porosis which, if of pathological degree, would negative the diagnosis of fatigue fracture.

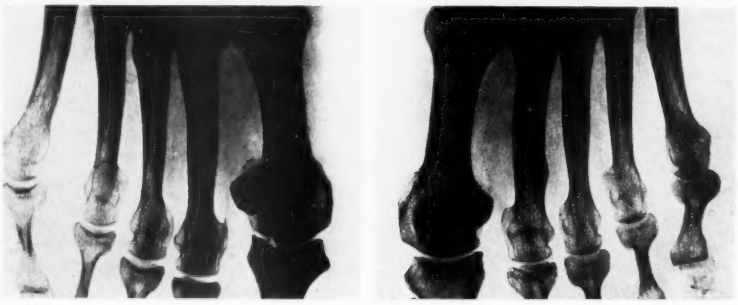

FIG. 10

Case 5. Radiographic evidence of old fatigue fracture of the second left metatarsal bone, of which a possible twenty-year-old history was given.

Comment—From this review, two groups of fatigue fracture of the lowest third of the fibula may be defined: 1) fractures in young male runners and skaters; 2) fractures in women of middle age or over, who have much to do on their feet. The example of the second group recorded by the writer (1940) was ascribed by Hartley (1943) to "spontaneous fracture due to age." But even if sixty-one be old, age is not a disease, and moreover there was a history of much activity and no obvious evidence of osteoporosis. The same is true of the cases here described, with the sole exception of the last which did show some generalised porosis, but apparently not of pathological degree so that there was little justification for the diagnosis of pathological fracture rather than that of fatigue fracture.

Clinical Features

A study of published and personal cases has yielded some knowledge of the clinical features of fatigue fracture of the lowest third of the fibula.

Age, sex, side affected, and activities of the patients are indicated in Table I (Section i). The natural grouping into young male runners and less young active women has already been mentioned. Four of twenty-one patients had bilateral fractures of the lowest third.

Site of fracture in the lowest third—The fracture was one and a half inches above the tip of the lateral malleolus in the five women observed by the author, whereas it occurred at a higher level in each of the two male patients. Study of other cases reported in the literature shows a similar distinction (Table II). It shows that most male patients (all the cross-country runners and skaters) suffered fractures two inches or more above the lateral malleolus, but that this was true of only one woman. If the difference in level is significant it may depend upon sex, age, or type of stress. Let it suffice at present to record that elderly, hard-pressed women tended to have fractures one and a half inches above the tip of the lateral malleolus through mainly cancellous bone at a level immediately below the interosseus ligament, whereas young male runners and skaters tended to have fractures at a higher level where the bone is slender but largely cortical. In this region the character of the bone, both as to thickness and composition, changes very rapidly with level (Fig. 11).

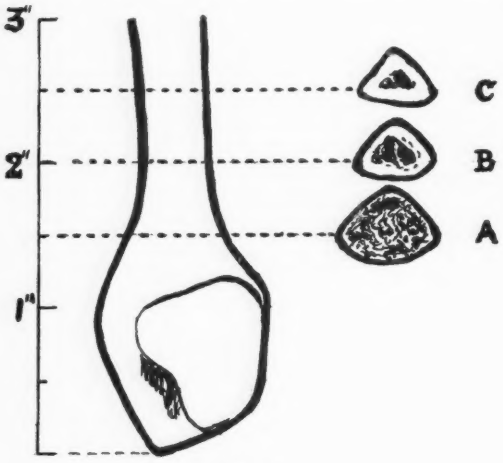

FIG. 11

Cross sections of the adult fibula at various distances from the tip of the lateral malleolus: A one and a half inches; B two inches; and C two and a half inches. The change in character within the short distance of half an inch between A and B is striking.

Symptoms—There has always been much activity involving repetition of the same resisted movement—walking, running, skating, and in one instance depression of a vibrating pedal. Usually the symptoms started during such activity but two patients here recorded first noticed pain on rising one morning. As a rule, customary activities are continued with difficulty for some days or weeks.

The first complaint has been *pain* and in the case of two patients a sensation of "stiffness." Occasionally the onset has been sudden, making one patient stumble and another fall. The site is described as the ankle, the lateral side of the ankle, the lateral malleolus, above the malleolus, or the outer part of the leg. There is relatively little information in the literature as to the characters of the pain, but in general it seems to be improved though not necessarily abolished by rest and to be increased by weight-bearing, especially weight-bearing with movement. It is sometimes worse on first resuming activity after a period of rest. As in fatigue fractures elsewhere, there is not only an absence of violence but also of any audible snap, so that no suspicion of fracture ever occurs to the patient.

Signs—*Localised tenderness* is usually present, but its absence was recorded specifically in three cases which were seen from three to eighteen days after the onset of symptoms (Hamilton and Finklestein 1944, Ingersoll 1943). *Localised swelling* was reported in all cases but five.

TABLE II

Author	Distance of Fatigue Fracture of the Lowest Third of the Fibula from the Tip of the Lateral Malleolus		
	Male	Female	Sex not stated
Grunert (1910)	?		
Finny (1924)	2 in.		
Detlefsen (1937 and 1941)		3 fingers breadth	About 6 cm.
Weaver and Francisco (1940)	3 cm.		
Burrows (1940 and 1948)	3¼ in. 2½ in.	1½ in. 1½ in. 1½ in. 1½ in. 1½ in.	
Ingersoll (1943) (Patients aet. 9 years)	2¼ in. 2¼ in. 3 in.		
Hamilton and Finklestein (1944)	About 1½ in.		
Richmond (1945)	2 in. 2 in.		
Ronald (1945)	2½ in.		
McPhee and Franklin (1946)	2 in. One-third length of fibula		

At first there is pitting or non-pitting oedema. This diminishes, and about three weeks from the onset of symptoms, a hard swelling of the tender area of the fibula becomes palpable. Slight *redness* has been reported twice (Hamilton and Finklestein 1944, McPhee and Franklin 1946). *Increased local cutaneous temperature* of slight degree has been recorded four times (McPhee and Franklin 1946, Burrows 1948). *Limp* was mentioned exceptionally. *Wasting* of the calf was found in one only of the writer's patients, the girth being diminished by half an inch. *Ankle and tarsal movements* are often undiminished. Most writers have failed to comment on movement but its freedom was remarked in six cases, from seven days to five weeks after the onset (Burrows 1940 and 1948, Detlefsen 1941, Hamilton and Finklestein 1944). Three patients in this series showed slight limitation of ankle movement but in no other known cases has limitation been recorded.

Radiographic Examination

During the first fifteen days or more no *radiographic change* may be discernible. On the other hand a *fracture line* may be evident after a week; sometimes none appears till the end of the second or third week. At first it is difficult to see, and even after four weeks it may be recognised only with the aid of a lens. A band of rarefaction may be seen at twelve to eighteen weeks. *Callus* has been observed in a child as early as the eighth day. In adults it appears during the third week, steadily consolidating and becoming organised at twelve to sixteen weeks when it forms a spindle-like thickening crossed by a less dense stripe at the site of fracture. At forty-seven weeks it may still be just possible to distinguish thickening. *A dense band* is often the first indication of the fracture site and almost the last to disappear. (See Figs. 1 and 6.) The plane of fracture is almost horizontal; slight deviations from this are not always in the same direction so that little can be concluded about the fracturing stresses.

Diagnosis

Since the condition is unfamiliar, and therefore unsuspected, it seems probable that it is often overlooked altogether. The freedom of joint movement and the site of tenderness help to distinguish it from sprains, but the absence of tenderness may be misleading. Radiographic examination must be the chief safeguard. In early suspected cases, if no abnormality is revealed with the aid of a lens the films should be repeated after three weeks. Competent interpretation is no less important than good radiography. Such diagnoses as osteomyelitis, tuberculosis, congenital syphilis, sarcoma and myositis ossificans have been made, and such errors may prove far more damaging than failure to recognise any lesion at all.

Treatment and Results

Provided that the exciting activity is discontinued, normal pursuits may be allowed with the comfort afforded by elastic adhesive bandage from the metatarsal heads to the upper calf. Physiotherapy is not required. With such treatment pain diminishes or disappears within a week, but tenderness may remain for as long as eight to sixteen weeks. Bone thickening persists still longer. It is difficult to be certain when the promoting activity may be resumed with safety; but too early resumption may cause recurrence of symptoms, or occasionally fatigue fracture of the opposite fibula or another bone.

FATIGUE FRACTURES IN THE UPPERMOST THIRD

Weaver and Francisco (1940) reported what were described as " pseudo-fractures," first in the lowest third of one fibula and then in the uppermost third of the other, in a student; but most recorded fractures at this higher level have been in soldiers. Hopfengärtner (1907) recorded military fractures of the uppermost third of the fibula in infantrymen during their first year of service. In twelve of the eighteen cases pain was first produced by jumping. The regimental records included twenty-one other cases, thirteen of them ascribed to jumping. Dreist (1909) reported fractures of the fibula at about the junction of the uppermost and middle thirds in two fusiliers during their first year of service. One sustained his fracture in landing from a vault and must be excluded because of the obvious trauma. The other noticed gradually increasing cramp-like pain during practice in attacking from the kneeling position. Radiography revealed an incomplete fracture which was attributed to strain from the nature of the exercise. Fractures in recruits reported by Grunert (1910) included one situated seven centimetres below the head of the fibula, sustained without apparent cause but interpreted as a torsion fracture. Scherf (1933) described fracture of the fibula at the junction of its uppermost and middle thirds, without direct injury and with little or no displacement, in nine artillery recruits who were required to carry out jumping exercises from a double-knees-bend position for about five minutes several times daily. Pain in the calf would start suddenly during the exercise. The author was uncertain whether the lesions were to be regarded as fractures or " transformation zones " (*umbauzonen*). Wachsmuth (1937a) emphasised jumping and repeated double-knee-bending as exhausting the " working efficiency of bone," the commonest effect being fracture of the fibula, usually at the junction of middle and uppermost thirds. Tibial march fracture, affecting mostly infantrymen and confined to the training period, might be accompanied by a corresponding fracture of the fibula. In the records of the German Army hospitals covering the year 1935–36, Asal (1936, 1937) found 590 cases ascribable to " overloading injury " and distributed as follows: metatarsals 488, tibia 70, femoral shaft 7, femoral neck 6, fibula 12, oscalcis 4, and pelvis 3. The fibular fractures, mostly affecting the junction of middle and uppermost third, occurred mainly in artillerymen and were ascribed to intensive gun exercises, particularly jumping on and off the limber over long periods. Wachsmuth also saw several such cases. Detlefsen (1937) described a case in a compulsory industrial worker. McPhee and Franklin (1946)

recorded one in a middle-distance runner. The frequent association with jumping is most striking. As the metatarsal fracture is the typical "march fracture," and the low fibular fracture is perhaps a running fracture, so the high fibular fracture appears to be typically a jump fracture.

It is interesting to compare fractures in the uppermost third of the fibula among infantry and artillery recruits with another series of fractures at the same site which constituted one of two typical leg injuries at a parachute school (Lord and Coutts 1944). These followed either parachute landings or platform jumps of four to six feet (Lord 1945). Pain was slight and patients often reported only after a delay of several days. Watson-Jones (1945) informed the writer that fifty-six fractures and dislocations sustained by a group of parachutists under training included "eleven cases in which a crack in the upper shaft of the fibula was suspected, or a fatigue fracture developed subsequently."

Former cases in artillery recruits and infantrymen, and more recent cases in parachutists, may be regarded as *either*: 1) traumatic fractures caused by jumps but often masked by painlessness; *or* 2) fatigue fractures; *or* 3) a combination of the two. Andreasen (1946) told the writer that the incidence of fracture of the fibula in its uppermost third amongst Indian parachute troops varied at different stages of their training but not in such a way as to suggest that fatigue was a material influence. Until further knowledge is available we cannot doubt the current view that high fibular fractures in infantry and artillery recruits are exclusively fatigue fractures, and that parachute fractures are exclusively traumatic fractures, but nevertheless the resemblance between these groups which each occur in young men undergoing severe training cannot be ignored, and it may be that trauma and fatigue both play a part in each group. A link would thus be forged between traumatic fracture due to a single adequate stress (abnormal *stress*), and fatigue fracture due to a summation of minimal stresses (abnormal *incidence*)—an interpretation which might be written thus:

	Stress	Incidence of stress
FATIGUE FRACTURE (*e.g.*, lowest third of fibula)	Minimal	+++++ etc.
RECRUITS' FRACTURE OFTEN ASSOCIATED WITH JUMPING (uppermost third of fibula)	+	++++ etc.
PARACHUTE SCHOOL FRACTURE (uppermost third of fibula)	++	+
TRAUMATIC FRACTURE	+++	Once

FATIGUE FRACTURE OF THE MIDDLE OF THE FIBULA

Siemens (1942) observed condensation and disintegration of bone structure in the middle of each fibula of a healthy boy aged sixteen months who complained of pain in the left leg when tired, and who limped. The right fibula showed periosteal new bone formation. Though the child was very lively the diagnosis of bilateral fatigue fracture of the fibula must, at this age, be accepted with reserve. Leveton (1946) described a fatigue fracture slightly above the middle of one fibula in a recruit aged nineteen years, and McPhee and Franklin (1946) reported a similar fracture just below the middle of the bone in a runner.

MULTIPLE FRACTURES

Multiple fractures naturally suggest general bone disease, which would exclude a diagnosis of fatigue fracture. The suspicion may prove false; multiple fractures sometimes occur without evident bone disease and they present all the typical characteristics of fatigue fractures and none of those of pathological fractures.

Fractures of both fibulae (at the same level or at different levels)—Bilateral low and high fatigue fractures have been mentioned. Low fracture of one fibula has been followed by high fracture of the other (Weaver and Francisco 1940).

Fractures of fibula and metatarsal—An infantry recruit, with bilateral low fatigue fracture of the fibula, later suffered a metatarsal march fracture (Hamilton and Finklestein 1944). Fatigue fracture of the third metatarsal bone in an infantry soldier under training was followed by a low fatigue fracture of the corresponding fibula (Richmond 1945).*

Except in their multiplicity these fractures differ in no respect from typical fatigue fractures. It is possible that although no pathological cause has been found there may be predisposition in some individuals. Poor neuro-muscular co-ordination may play a part, as suggested by the observation of non-commissioned officers that recruits who developed such fractures had been awkward from the first (Wachsmuth 1937b). Moreover, Case 5 of this series shows that otherwise typical fatigue fractures at multiple sites (fibula and metatarsal) may in fact be associated with slight non-pathological osteoporosis.

* Wachsmuth (1937b) mentioned an infantry recruit with fractures of one tibia and fibula between middle and uppermost thirds, who was found to have an old united high fracture of each fibula; it is possible, however, that the recent fractures were traumatic. High fracture of the fibula sometimes complicates fatigue fracture of the corresponding tibia, but it is possible that the fibular fracture is a traumatic complication of displacement of the tibial fracture, as in a case of Hartley (1942).

SUMMARY

1. A hope expressed in 1940, that further cases of spontaneous fracture of the lowest third of the apparently normal fibula would be described, has been fulfilled. The literature is here reviewed. Five further personal cases are added.

2. The clinical and radiographic features, diagnosis, treatment and results are considered in the light of the information so far available. Special note is made of misleading freedom of ankle and tarsal movements and the occasional absence of tenderness.

3. It is established that fractures of the lowest third occur particularly in two groups of subjects: 1) young male runners and skaters; 2) active and hard-pressed women of middle age and over.

4. In male runners and skaters the fracture usually occurs through slender, mainly cortical bone, two inches or more above the tip of the lateral malleolus; in middle-aged women the fracture is usually distal to the interosseous ligament through thicker, mainly cancellous bone, one and a half inches from the tip of the lateral malleolus.

5. The most convenient name for both groups of fractures in the lowest third is *low fatigue fracture of the fibula*.

6. A review of the literature of fatigue fracture of the uppermost third of the fibula shows that it is very often precipitated by jumping. The most convenient name for it is *high fatigue fracture of the fibula*.

7. Like all clinical classifications this distinction between low and high fractures has exceptions (a low fracture of one fibula in a runner was followed later by a high fracture of the other; most military fractures were high, but a few may have occurred at other levels).

8. Fatigue fracture of the fibula, high or low, may be bilateral.

9. A fracture similarly situated to the high fatigue fracture of the fibula has been frequent in parachute schools. It is a speculative possibility that military and parachutist fractures of the upper third of the fibula indicate the link between true fatigue fractures (as exemplified by march fractures with minimal trauma often repeated) and purely traumatic fractures (with adequate trauma applied once only).

Thanks are due to Major Lord, U.S.A.M.C., the late W. Rowley Bristow, Professor A. I. Andreasen and Sir Reginald Watson-Jones for information given; and to Dr W. R. Bett and the Librarian of the Royal Society of Medicine for their help with the literature. Case 3 was referred to me by my colleague, Mr W. D. Coltart.

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THE MANAGEMENT OF ACUTE CIRCULATORY FAILURE IN AN INJURED LIMB

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Paper read in Manchester at the meeting of the British Orthopaedic Association, October 1947

Sudden arrest of the circulation in a limb is a catastrophe of particular interest to the orthopaedic surgeon because, apart from embolism and arterial disease, this emergency is almost confined to the vascular complications of fractures and dislocations. Crushing injuries of a limb, and accidents after the use and abuse of tourniquets are rare.

The fractures in which the circulation is most in peril are those of the lower end of the humerus, the upper parts of both bones of the forearm, the shaft and lower end of the femur, and the upper two-thirds of the leg bones. Actual gangrene complicating fractures in civil life occurs with greatest frequency when both anterior and posterior tibial arteries are injured as the result of high fracture of the tibial shaft, but arterial injury as such is found most

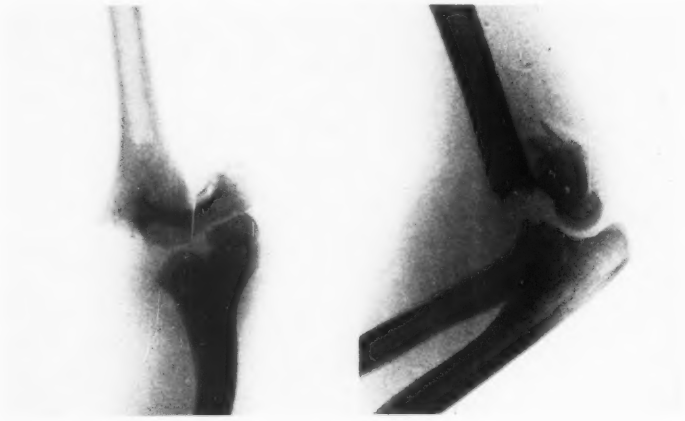

Fig. 1

Supracondylar fracture lower end of humerus in a child of eight years with loss of radial pulse but no ischaemia of hand or forearm. There was no nerve lesion.

often in association with the much more common supracondylar fracture of the lower end of the humerus (Fig. 1). The combination of fracture with vascular obstruction is nearly always the result of severe injury, and the dice are loaded against the surgeon who must work upon the badly damaged ischaemic limb of a patient with little reserve of strength. Yet, surgical treatment must be prompt and radical, for acute arterial occlusion carries real danger to life as well as to the limb itself.

PRE-OPERATIVE INVESTIGATION OF TRAUMATIC ISCHAEMIA

Diagnosis—It is unhappily true that complete arrest of the arterial circulation of an injured limb is seldom recognised until too late. Moreover, even when the condition is diagnosed early enough for intervention to be useful, effective action is too often delayed by an attitude of wishful thinking. To be sure, the clinical features of acute ischaemia are usually plain and striking enough to call for immediate action. The mnemonic list of "pain, pallor, paralysis, and pulselessness" is perhaps an over-simplification, but it needs little elaboration.

All four are usually present in greater or less degree. *Pain* is perhaps the least constant. It may sometimes be absent, and it may vary from unpleasant numbness to real agony; but any pain, severe or slight, in the distal extremity of an injured limb must call for assessment of the circulation. *Pallor* is only one of a series of colour changes which may later pass from cadaveric grey-white through stages of irregular cyanosis to the black of frank gangrene. Pallor is, however, the first change and therefore the most important.

FIG. 2

Closed fracture shaft of right tibia in a man aged twenty-four years. Arteriogram shows occlusion of both tibial vessels almost to the popliteal bifurcation which proved to be due to contusion at level of fracture with thrombosis. Gangrene occurred. Below-knee amputation was successfully performed after lumbar sympathectomy.

FIG. 3

Closed fracture shaft of left femur in a man aged twenty-nine years with occlusion of superficial femoral artery by compression against the roof of Hunter's canal by a large haematoma. Signs of ischaemia developed seventeen hours after injury when the systolic blood pressure was 70 mms. The patient died.

Paralysis, both motor and sensory, takes a short while to be established and may be confused with the effects of nerve injury, but *absence of the distal pulses* is immediate and invariable. It is a sign more difficult to elicit in the lower limb than in the upper limb. Moreover absent pulses may not of necessity signify inadequate limb circulation. But the fact remains that the distal pulses must be sought, and that their absence may not be discounted lightly in any injured limb.

Despite some variability, it is remarkable how constant these signs are, and there is seldom any real doubt as to the adequacy of blood supply to a hand or foot. The *site* of

arterial block is usually indicated fairly clearly by the site of the fracture. The precise level can be determined by examination with an oscillometer or, at the time of operation, by arteriography. If there is doubt, it is usually true that the upper limit of block is a good deal higher than the surgeon is inclined to expect (Fig. 2).

Pathology—Apart from complete or incomplete division due to laceration by the fragments of a fracture, the arteries of an injured limb may sustain a variety of lesions in continuity. The most important are compression, intra-mural rupture, and contusion. Complete rupture

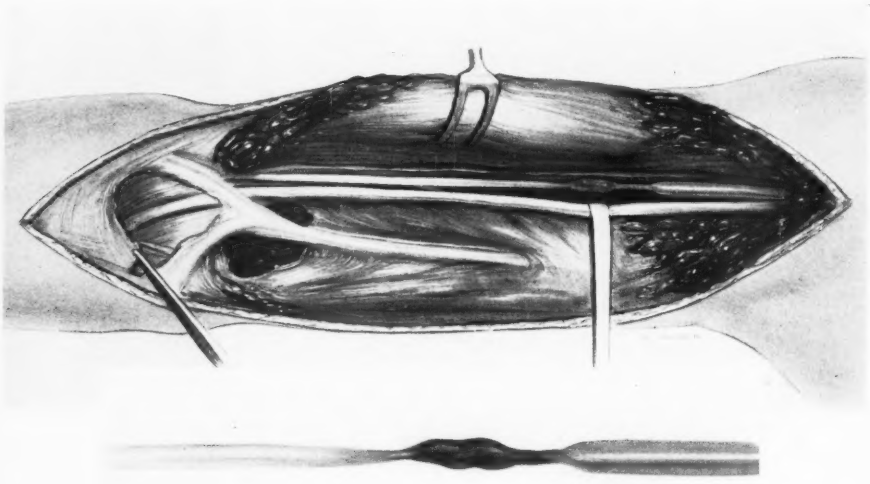

FIG. 4

Intra-mural rupture of the brachial artery in a man aged forty-five years as seen at operation six hours after injury due to crushing by machinery. The lesion was accompanied by widespread arterial spasm which relaxed after excision of the damaged portion of the artery.

can follow traction injuries and has occurred in this way at the upper end of the popliteal artery following hyperextension of the knee (Wakeley and Reid 1932), but any form of rupture which produces an arterial haematoma or false aneurysm is extremely rare in civilian injuries.

Compression may be due to simple stretching across a displaced fragment, or to actual incarceration of the vessel between two fragments of a fracture. Very seldom, far more seldom than is often taught, the compressing agent may be a haematoma under tight deep fascia, but it will be clear that a haematoma with sufficient tension to occlude a main artery must have been formed when the arterial pressure was considerably higher than at the time of compression. Such occlusion by the pressure of a haematoma can occur therefore only some time after the injury, in a patient whose fallen blood pressure indicates even more grave problems than those of limb ischaemia (Fig. 3).

Intra-mural rupture (Figs. 4 and 5), the outer coat of the vessel being intact, occurs in traction injuries and crushing injuries. It is, of course, the usual result of ligature in continuity.

Contusion, the commonest injury, may be severe or slight, but either degree can arrest the blood stream immediately by arterial spasm (Fig. 6) or later by thrombosis of the contused segment (Fig. 7).

Traumatic arterial spasm may accompany any form of arterial injury and produce widespread block which magnifies the effect of mechanical obstruction. It may produce extensive

occlusion of an artery which shows no evidence of damage to the naked eye and which, indeed, may have received no more than a " near miss." Nevertheless conduction of blood may cease, just as conduction of nerve-impulses may cease in injuries which have produced no apparent damage to the nerve trunk. The cause, nature, and propagation of spasm are obscure, but it must be emphasised that ischaemia from spasm of an apparently undamaged artery is clinically indistinguishable from that due to mechanical obstruction, and that spasm and mechanical obstruction are usually present together. There is nothing essentially benign in traumatic arterial spasm, and to hope that an ischaemic limb is suffering " only " from spasm which " will probably relax " is to court disaster.

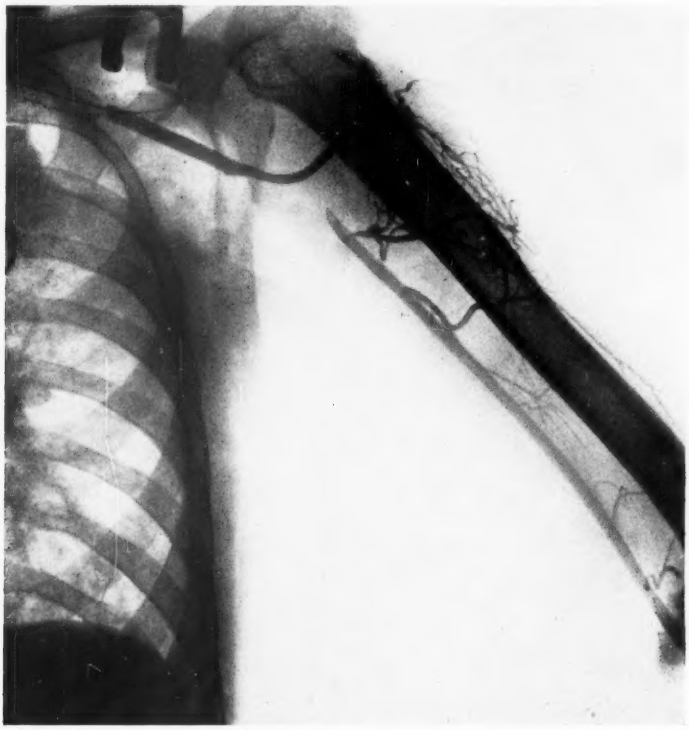

FIG. 5

Arteriogram eight weeks after intra-mural rupture of the brachial artery at its extreme upper end due to forced mobilisation of the stiff shoulder of a man aged thirty-three years. There was a history of immediate pallor and paralysis of the limb. Recovery of paralysis was very incomplete and there was still anaesthesia of the hand.

Investigation of a case of traumatic ischaemia—However clear his idea of the pathology may be, the surgeon still needs all possible information before proceeding to treatment. Examination of the limb *and of the patient* must be complete. He must know the blood-pressure, the haemoglobin content of the blood, the points at which pulses are present and those at which they are not. Complete radiographic study of the bone injury is essential. Oscillometry, skin thermometry and even the taking of muscle temperatures (Adrian and Watts, 1923) may give useful help. The coagulation time should be known in case an anti-coagulant may be needed later. Treatment is urgent, but not so urgent that investigation can be scamped.

TREATMENT OF TRAUMATIC ISCHAEMIA

Principles of treatment—There are four principles of treatment, the first two being applicable to every case:

1) Removal of any possible external pressure;
2) Restoration of the patient's general condition.

If these measures fail to relieve the circulation in the injured limb, and they usually do fail, the surgeon must proceed to:

3) Operative exposure of the site of obstruction, with appropriate action, followed by
4) Post-operative management which is perhaps no less important than the operation itself.

Pre-operative measures—The removal of any possible external pressure means that *all* encircling bandages and plaster casts must be removed and all traction apparatus relaxed, even if the dressings and extensions do not appear to be tight. The limb should be placed in the most comfortable position, slung on a Thomas splint, or simply supported on pillows, while general treatment is pursued. An intravenous saline drip infusion, or in the case of lower limb injuries in adults a blood transfusion, is usually required. This is particularly true because if operative intervention becomes necessary the one thing that must never happen is a fall of blood-pressure during or immediately after operation. An alarmed and urgent call from the anaesthetist for blood transfusion at the end of the operation usually implies in itself that the outlook for recovery of the limb is poor.

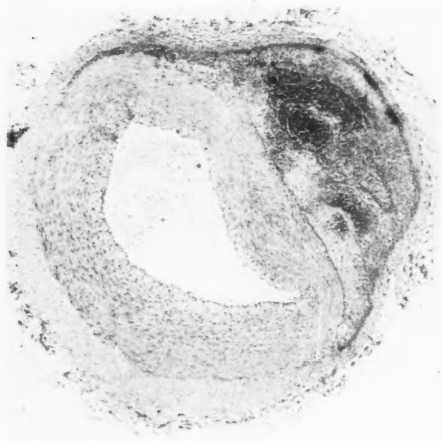

FIG. 6

Contusion of brachial artery with haematoma of the wall but no thrombosis, due to supracondylar fracture of humerus in a boy aged seven years. The widespread arterial spasm relaxed after excision of the contused portion. (Reproduced by kind permission from the British Journal of Surgery 1940, **28**, 252.)

The operation—Good general anaesthesia is essential. The theoretical advantages of spinal analgesia in ischaemia of the lower limb are usually outweighed by the dangers. A case can be stated for local anaesthesia, but not a very convincing one.

As soon as anaesthesia is induced, one and only one attempt should be made to reduce the fracture by manipulation. Manipulation may release a trapped artery and therefore lead to full restoration of the circulation and occasionally to return of the distal pulse, but it is more likely to improve the circulation, perhaps to a safe level, by relieving pressure on collateral vessels (Figs. 8 and 9). It must be remembered, however, that repeated manipulation of displaced fractures is more likely to cause arterial injury than to remedy it. Accordingly, no more than one attempt should be made. No time should be wasted in attempting mechanical reduction or applying skeletal traction, and if one manipulation fails to improve the circulation open operation is at once indicated.

I believe it is wise first to expose part of the main artery a short distance above the obstruction, to pass a loose rubber sling under it in case quick control should be needed, and then to perform an arteriogram. The technique of arteriography is described by Learmonth (1944). The contrast medium should be a diodone preparation, my own preference being for " Pyelosil " (Glaxo) in 50 per cent. solution for adults, and in 35 per cent. solution for the smaller arteries of children, which can be entered only by needles too fine to pass the

more viscous 50 per cent. solution. The picture so obtained may give great help in deciding the future plan of the operation.

The next step is usually to make a generous exposure of the centre of occlusion unless such exploration is clearly too late, or cannot be performed without damaging collateral vessels. The decision as to whether or not to explore, calls for experienced judgment, but it may be stated as a general guide that little good can come from attempts to relieve total ischaemia of twelve hours' standing, and that even this is a generous estimate. Furthermore, relief of long-standing ischaemia is not always desirable. I have seen restoration of the circulation after twenty-four hours' obstruction kill the patient by flooding his general circulation with the products of tissue damage in the infarcted limb. My colleague A. N. Guthkelch (1947) has seen severe renal changes after successful release of no more than a twelve-hour block of the common femoral artery.

The time factor is of particular importance in the case of tourniquet accidents. It is to be regretted that, in the present state of our knowledge, the best advice that can be given is that a tourniquet which has occluded a thigh for six or more hours should not be removed at all, but should be amputated with the limb. It is to be hoped that work now being undertaken will lead to a happier solution to this grave problem.

The danger of damaging the collateral circulation arises particularly in obstructions of the upper parts of *both* anterior and posterior tibial vessels. Obstructions at this level are seldom amenable to direct surgical relief, and the anatomy of the collateral vessels is such that it is doubtful whether these vessels should ever be explored for acute traumatic occlusion. Here the value of an arteriogram is manifest (Fig. 2). Other things being equal, it may be wise to explore a blocked popliteal artery, but if radiographs show that the block is at a lower level the surgeon will be wise to hold his hand.

FIG. 7

Contusion with thrombosis in the popliteal artery of a man aged nineteen years who sustained a hyperextension injury of the knee joint at football two months before this arteriogram was made. Despite rich collateral circulation, ischaemia persisted and produced classical contracture of the calf muscles.

Treatment of the damaged artery—When there is no contra-indication, the occluded vessel is released and widely exposed. Reparable lesions should be repaired. Lateral suture of a torn artery by the Carrel (1907) technique, though seldom possible, may save an important

channel and should not be followed by thrombosis even if anticoagulants are not employed. On the other hand end-to-end suture of a complete tear, as distinct from an incised wound, is never practicable.

Irreparable damage is a more common finding. The badly contused or irregularly lacerated vessel, or the artery which shows evidence of intra-mural rupture, should be treated by resection of the damaged portion, as advocated by Makins (1919) nearly thirty years ago. This much debated operation of *arterectomy* can do no harm in injuries of this nature, and will at least prevent spreading thrombosis and secondary haemorrhage which is more than can be said for ligation in continuity, an operation which should never be performed.

FIG. 10

Arteriogram four and a half months after arterectomy of a damaged brachial artery in a man aged twenty-five years. Despite free collateral circulation through the profunda and other vessels, the hand remained partly anaesthetic. The proximal stump of the brachial artery is indicated at A.

Treatment of arterial spasm—The artery which is found to be intact and apparently undamaged, but tightly occluded by widespread spasm, presents a difficult problem. All possible conservative measures for the relief of spasm should first be attempted. Wide mobilisation of the vessel has had success. If it fails, procain should be injected around the vessel and even into the lumen. The other limbs should be heated by immersion in hot water. Arterectomy should not be considered unless there is a visible local lesion. Above all, arterectomy is to be avoided if the vessel is in fact transmitting any blood.

Though no one doubts the wisdom of arterectomy for irreparable local lesions, there is much dispute as to whether the procedure is ever indicated for spasm alone. There are no reported cases in which arterectomy has relieved spasm of more than twelve hours' duration, but earlier operations (within seven hours of injury) have caused, or have at least been followed by, striking relief of spasm and cure of ischaemia (Griffiths 1940). Whether or not a reflex arc is broken, and whether or not the trans-section and release of smooth muscle influences the relaxation, early arterectomy has not only prevented spreading thrombosis or embolism from the site of damage, but has apparently also relieved spasm. Arterectomy in the adult limb does, however, tend to be followed by unpleasant and persistent symptoms such as digital anaesthesia or intermittent muscle cramp, and the conclusion must be drawn that the procedure should be reserved for vessels irreparably damaged, and vessels which are certainly empty and will never again transmit blood. In this way, at least no harm will be done. There is some hope, too, that further experimental work with cannulation or vein grafting may result in the development of a technique by which arterectomy can be followed by restoration of the channel. (Mustard 1945, 1946.)

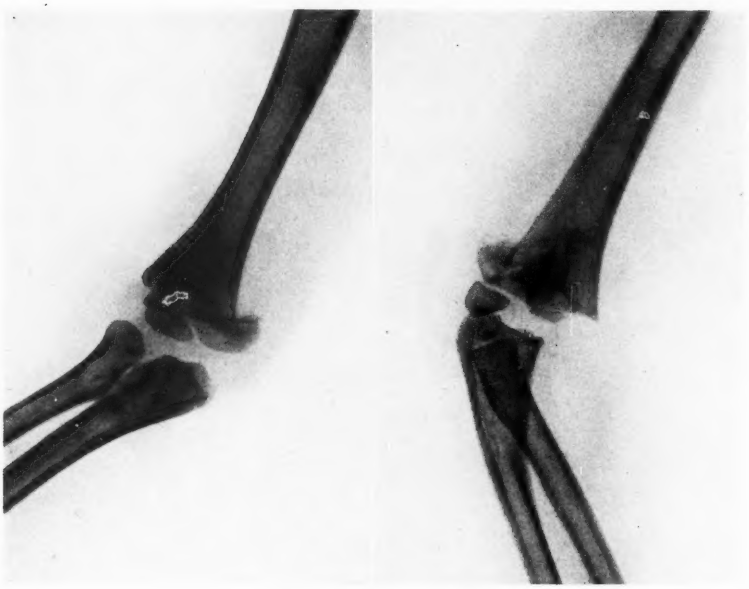

FIG. 8

Supracondylar fracture of the humerus in a child aged six years with absence of the radial pulse and pallor of the hand.

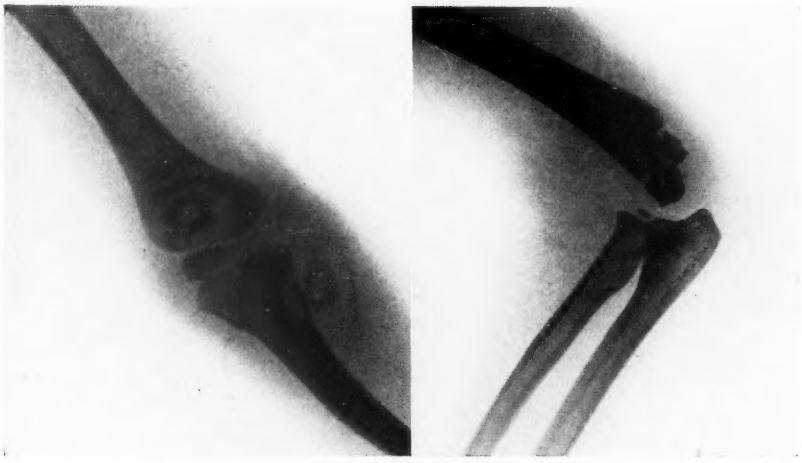

FIG. 9

Manipulative reduction of the fracture in Fig. 8 succeeded in restoring the circulation in the hand, but the radial pulse did not return. Presumably improvement resulted from relief of pressure on collateral vessels.

SYMPATHECTOMY

If arterectomy is deemed necessary, or if operation results in anything short of full restoration of the circulation, the sympathetic supply of the limb should be interrupted. In the lower limb, if the condition of the patient permits, this is best done by lumbar ganglionectomy. With good modern anaesthesia this operation is neither difficult, lengthy, nor dangerous, and it should be part of the orthopaedic surgeon's standard repertoire. In the upper limb, however, adequate pre-ganglionic section is a different matter, and should not be attempted by occasional workers in that field.

However inexperienced a surgeon may be in operative sympathectomy, he is capable of paravertebral injection of procain. Such injections, repeated as necessary, will usually suffice and may indeed be all that is permitted by the condition of the patient. Simple methods of blocking the sympathetic supply to both upper and lower limbs are described in a Medical Research Council Memorandum on Arterial Injuries (1944), and by Gage and Ochsner (1940), White and Smithwick (1942), Caldwell, Broderick, and Rose (1946), etc.

As to the value of sympathetic block, operative or chemical, there can no longer be dispute. The experience of many surgeons in vascular centres in the recent war (Boyd 1946, Mason and Giddings 1945) has reinforced the experimental deductions of Barnes and Trueta (1942) and has not supported the fears of Cohen (1944) and Siddons (1945) that sympathetic block may even be harmful.

DRUGS

Vaso-dilator drugs have been disappointing. Eupaverine (Denk 1934) is probably useful; papaverine is certainly not. Tetra-ethyl ammonium bromide (*vide* Berry *et al.* 1946) is still on trial. It promises well as a means of paralysing the sympathetic, but it has potential dangers.

Heparin and other anti-coagulants may be indicated to prevent or limit thrombosis. Alkalies are of great value, and should always be given as a protection against renal failure. The only other useful drugs are morphine and other sedatives, which should be used freely.

POST-OPERATIVE CARE

The first few post-operative hours are critical. All necessary measures must be taken to maintain an adequate systemic blood-pressure, for a good head of pressure will obviously help to force blood into an injured limb. Alkalies should be continued and since ischaemia of tissues obviously predisposes to infection, penicillin should be given as a routine. The limb should be slung on some " open " form of splintage, controlled but not compressed. It should be elevated just above the level of the heart unless the patient finds greater comfort just below that level. It should be kept cold, though not iced, in the shade before an open window. An electric fan is needed in hot weather. The rest of the body should be heated, even until the temperature rises to 100 or 101 degrees Fahrenheit. An electric blanket is useful.

Drugs play little part in the post-operative care. Sound sleep and freedom from pain should be obtained by the liberal use of morphia and of sedatives. Nicotine is a vaso-constrictor and there seems reasonable evidence for the prohibition of smoking. It is, however, consoling to note that alcohol is a vaso-dilator.

SUMMARY

Ischaemia threatening an injured limb gives rise to the syndrome of pain, pallor, paralysis, and pulselessness. It is due to arterial injury by laceration, compression, intra-mural rupture or contusion, or to arterial spasm with or without demonstrable local arterial damage. The differentiation of spasm without local injury from organic obstruction is not possible by clinical methods.

The suggested plan of treatment and of management is:

1. General systemic investigation (blood-pressure, blood-count, coagulation time, etc.).
2. Removal of all external pressure.
3. Resuscitation.
4. Direct attempt to relieve the obstruction by operation.
5. Post-operative care.

The operative procedure recommended is:

1. Manipulative reduction of the fracture if possible.
2. Proximal control of the artery.
3. Arteriography.
4. Exposure of the occluded artery (unless contra-indicated by time factors and by the anatomy of the collateral circulation), liberation and mobilisation of the vessel, repair by suture where such is necessary and possible, and arterectomy only for irreparable local damage.
5. The provision of sympathetic block by injection or by sympathectomy.

The important elements of post-operative care are:

1. To maintain the blood-pressure while cooling the limb and heating the patient.

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THE CLINICAL DIAGNOSIS OF FAT EMBOLISM

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Three conditions which were responsible for high mortality in the recent war and for which there is not yet a fully agreed pathological explanation, or a specific form of treatment, are the crush syndrome, traumatic uraemia, and fat embolism. It is generally believed that fat embolism occurs more often in war wounds than in civilian injuries; but it is hoped to show in this paper not only that the frequency of the condition is under-estimated, and that recognition is comparatively easy provided only that the clinician is on the look out for it, but that it occurs in civilian injuries much more often than is thought. Of patients under my care in military surgical units during 1945 there were seven with fat embolism and, although the majority of all traumatic cases seen at that time were battle casualties, five of these seven were due to road accidents. The details are recorded in Table I.

TABLE I

ANALYSIS OF SEVEN CASES OF FAT EMBOLISM

Case No.	Site of injury	Type of injury	Onset	Petechial rash	Mental change	Fundus	Surgical therapy	Result	Duration
1	Femur	Road accident	Under 24 hours	Chest, neck, conjunctiva	Deep coma —three attacks	?	—	Died	14 days
2	Both tibiae	Road accident	2nd day	Chest, back, abdomen, conjunctiva	Deep coma	?	—	Died	3 days
3	Femur	Road accident	3rd day	Conjunctiva, few on neck	Slight "wandering"	?	—	Recovered	—
4	Femur	Road accident	Under 24 hours	Chest, neck, abdomen, conjunctiva	Delirium— two attacks	Haemorrhage Oedema	Ligation of vein	Recovered	—
5	Femur	Shell wound	2nd day	neck, conjunctiva	Deep coma	Haemorrhage Oedema	—	Died	5 days
6	Right femur, left tibia	Road accident	2nd day	Conjunctiva, few on neck later	Deep coma	Haemorrhage Oedema	Ligation of vein	Died	7 days
7	Tibia	Mine injury	2nd day	Chest, neck, conjunctiva	Deep coma	Haemorrhage	—	Died	9 days

During a three-month period after the cessation of hostilities, at two military hospitals in Germany, eighty-nine major injuries of the long bones were admitted. These included twenty-three fractures of the femur and forty-six fractures of the tibia, the majority of which were due to road accidents. Five cases of fat embolism were diagnosed; four were due to road accidents and the other to an accidental gun-shot wound of the lower end of the femur. Three proved fatal.

Wilson and Salisbury (1944) reporting on 1000 consecutive battle casualties, including 119 fractures of one or more long bones, found that there was clinical evidence of fat embolism in eight cases of which six were fatal—a mortality rate of 75 per cent., or for all fractures

of long bones 5 per cent. Vance (1931), working in New York, reported that post-mortem examination of fifty-nine accident cases with fractures of long bones showed that 20 per cent. had severe fat embolism and 55 per cent. had moderate or slight fat embolism; that is to say that three-quarters of these civilian accidents with fractures of a long bone which proved fatal showed some degree of fat embolism. Robb Smith (1941), working in Oxford, reported that of 125 deaths due to accident, forty-one (33 per cent.) showed gross fat emboli at post-mortem examination. In his paper he stated: "There is little doubt that many of the complications of injury such as traumatic pneumonia, delirium, delayed shock and blast are in reality fat embolism." To this list might be added concussion. The patient who is admitted with a fractured femur and a bruised or even fractured skull, and who lapses into delirium and coma, may easily be classified as a fatality due to head injury. Of ninety-three post-mortem examinations performed by Mavor in the Middle East in 1942 of which forty had a fracture of one or more long bones, seven deaths were due to fat embolism. These figures are given as examples of accumulating evidence that: 1) fat embolism is a relatively common complication in fractures of the long bones; 2) the diagnosis is made less often before death than after; 3) it occurs almost as frequently in civilian accidents as in battle wounds.

Fat embolism usually occurs during the third and fourth decades and is more common in men than in women. It follows that in time of war, military services include a preponderance of those who are prone to this complication, whereas the civilian population is correspondingly depleted. Furthermore the gravity of wounds, and the long journeys down lines of communication over rough roads or tracks, may account for a higher incidence in war-time. But there is no evidence of any specific etiological factor relating to missile wounds. During peace-time severe accidents are less frequent and often they are treated by junior members of the hospital staff. The surgeon himself seldom sees the patient with a fractured femur who is admitted to a small remote hospital during the week-end rush on the roads and is dead within forty-eight hours; he is simply informed that an accident case with head injury died after admission from secondary shock, concussion, or cerebral haemorrhage. Fat embolism can so easily be mistaken, not only by the clinician but by the pathologist, that unless the possibility is kept constantly in mind the correct diagnosis will be missed. Many a petechia has blushed unseen by the surgeon who failed to pull back the bed clothes, examine the base of the neck, and inspect the conjunctival sac.

PATHOLOGY

Fat emboli within the capillaries can best be found by the method described by Robb Smith in which a snippet of lung is cleared with potassium hydroxide. Microscopically there are petechial haemorrhages which may be distributed widely but are sometimes less obvious. Horizontal sections of the brain generally show a remarkable picture of densely packed petechiae throughout the white matter but none in the grey matter (Fig. 1).

Excellent surveys of the literature by Scuderi (1934) and Grossloss (1935) indicate the mass of experimental and pathological evidence which has accumulated, but there is still no proof as to the source of the fat. It may arise from the wound area; it may arise as an abnormality of general metabolic processes; or it may be that there is both embolism from the wound and general metabolic disturbance. Busch (1866) injected vermillion into the medulla of a long bone, fractured it, and after five minutes found pulmonary fat emboli stained with the dye. He showed that nearly all the fat travelled through the veins and not lymphatics. Gauss (1924) demonstrated that when veins inside the rigid walls of a bone are damaged they tend to remain open whereas those in soft tissue do not. At least one observer has found that blood taken from the femoral vein on the side of injury has a higher fat content than on the opposite side. Reiner (1907) reported good results from the application of a tourniquet, insertion above the tourniquet of a cannula into the femoral vein by way of the internal saphenous vein, release of the tourniquet, and drainage of the excess of fat.

On the other hand Lehman and Moore (1927) showed that the femur contained only a maximum of 65 c.c. of fat whereas the lethal dose, if man can be considered to show the same tolerance as the dog, is 120 c.c. Groendahl (1911) found that ligation of the femoral vein and removal of the lymphatic inguinal glands on the side of injury did not prevent fat embolism. Vance reported post-mortem evidence of fat embolism not only in 75 per cent. of fatal fractures of long bones but also in 12 to 14 per cent. of non-traumatic deaths. Catsaras (1920) found fat embolism in the lungs of 18 per cent. of sixty-seven patients dying from post-influenzal pneumonia. Fat embolism has been reported in a case of concussion without fracture (Ribbert 1894); in diabetes mellitus (Fitz 1881, Machlis 1924); in pulmonary tuberculosis (Heitzman); in chronic osteomyelitis (Field 1913); and after orthopaedic operations (Coolidge 1901, Codivilla 1910, Turner 1913, Schwamm 1926).

FIG. 1

Horizontal section of brain showing many petechiae in the white matter but none in the grey matter. (By courtesy of the British Journal of Surgery.)

Whatever be the origin of the fat globules the view that their harmful action in the tissues is due to simple embolism and obstruction of blood-vessels cannot be accepted. Histological evidence shows that the fat, in disintegrating, causes destruction of the wall of the vessel with perivascular oedema and haemorrhage. A globule of inert fat would not be expected to act in this way. Hirsch (1941) pointed out that the reaction of tissues to unhydrolised fat was similar to that of inert oil-like liquid petrolatum. On the other hand hydrolysis of fats liberates fatty acids and other chemical bodies which presumably are responsible for destruction of the wall of the vessel and for pathological changes in the perivascular tissues.

CLINICAL FEATURES

Scuderi (1934) concluded: " The symptoms and signs are very difficult to elicit clinically. The symptoms are indicative only in a general way and are not at all conclusive. The signs are of more value but they are not conclusive enough." These observations were unduly pessimistic. Only the lesser degrees of fat embolism, not endangering life, lack symptoms or signs to aid the clinician. In serious cases there are always signs, sometimes extensive

and obvious, but often unobtrusive and not recognised unless carefully sought. If the clinician knows what to look for, and where to look for it, the diagnosis is easy.

Clinical history—The typical clinical picture is that of a man in the third or fourth decade who in consequence of a road accident has sustained a compound fracture of the femur and is admitted to hospital, perhaps after a long and rough journey with the limb improperly immobilised, suffering a considerable degree of shock. The response to intravenous transfusion of blood or plasma is good; within three or four hours the blood-pressure and pulse rate are normal, and the patient is perfectly conscious and co-operative. Operative treatment of the wound and reduction of the fracture under intravenous and inhalational anaesthesia is arranged. An hour after operation the pulse is of good volume, the blood-pressure is maintained, and the general condition is satisfactory. The fact that consciousness has not yet been regained calls for no comment. Some hours later, however, when it is found that the patient is still deeply unconscious, the possibility of fat embolism is brought to the fore. The patient may remain in this state and die quite soon, in which case the anaesthetist is liable to be blamed for a poorly administered anaesthetic, or the patient be blamed for an idiosyncracy to the particular drug used. In such cases the anaesthetist should demand a post-mortem examination and a special search for evidence of fat embolism.

Other degrees of cerebral disturbance may occur. The patient is often unco-operative--refusing to eat food, refusing to use the urine flask, constantly calling for the nurse, repeatedly ringing his bell, and generally making himself a nuisance. This attitude, which is of important clinical significance, may well be overlooked in so far as it is not reported to the surgeon by the nurse. Other patients are restless or violent, perhaps needing two orderlies to keep them in bed. They may lie in a semi-conscious, delirious, stuporous, or comatose state with pale sweating skin and stertorous breathing. A patient may exhibit varying degrees of disturbance at different times, first sinking into deep coma, then regaining full consciousness, only to be plunged once more into coma when a fresh crop of emboli lodge in the brain. Alternate elevation and depression of consciousness due to successive showers of emboli is very typical and accounts for the association of fresh, recent, and old healing lesions of the brain which is often found at post-mortem examination.

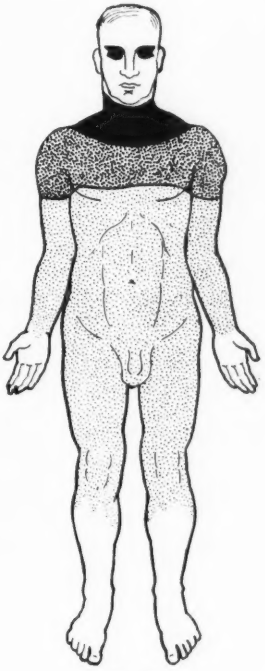

FIG. 2

Petechial haemorrhages are most common in the supraclavicular regions and the conjuctivae.

General clinical signs—*Pulse*—The pulse rate is slightly raised; the volume is good. The veins are dilated and there is some dilatation of the right side of the heart. Killian (1931) reported dilatation of the heart in ten cases. *Blood-pressure*—The blood-pressure is normal or raised, thus at once ruling out the possibility of severe secondary shock. *Respiration*—There is respiratory embarrassment, raised respiration rate, frothing at the mouth, cyanosis, deep stertorous breathing, and often Cheyne-Stokes breathing in the terminal stages. There may be pain in the chest with signs of oedema and consolidation at the bases. *Temperature*—The temperature is high; a reading of 102–105 degrees is usual and it may go even higher in the terminal stages. *Central nervous system*—The signs are usually of a general nature with changes in the conscious state, muscle rigidity, loss of sphincter control, convulsions, and sometimes Jacksonian fits. Local signs such as facial paralysis (Aberle 1907), absent ankle jerks and positive Babinski response (Watson 1937) have been reported.

Petechial haemorrhages—Petechiae may be abundant or sparse; their number has no relation to the prognosis. When few, they are to be found at the base of the neck and in the conjuctival sac; they must be sought carefully in a good light or they may easily be missed. In the conjuctiva they occur chiefly in the inferior half; the lower lid must be pulled down to reveal them (Fig. 3). Even in a case of grave prognosis there may be no more than one or two conjunctival petechiae. The other common site is the posterior triangle of the neck. When abundant they extend over the chest and abdomen and sometimes to the limbs. They are most thickly distributed over the neck, deltoid region, anterior aspect of the axilla and chest (Fig. 2). They may appear in recurrent crops which synchronise with the attacks of coma.

FIG. 3

Three petechiae seen on the inner side of the lower lid.

The fundus—Globules of fat coursing through the retinal vessels were first seen by Muller (1860) and have been reported in a case of diabetes mellitus by Bantin (1926); these globules disappeared after the administration of insulin. Oppenheimer (1929) reported retinal fat globules in a case of fat embolism. This sign, however, is not the essential finding in fat embolism. More typical are the pathological changes in the substance of the retina itself, described recently by McArdle (1946). They consist of yellow-white glistening patches of perivascular oedema, or patches of perivascular haemorrhage, occurring along the line of the vessels (Fig. 4). When searching for them each vessel should be traced from the disc to the periphery. It was not until the fourth case of this series came under my care that I learned of these changes from McArdle, but in the last four cases they were deliberately sought and were found in every one. Wilson and Salisbury (1944) were unable to find fat globules in the vessels but they make no mention of the retinal changes just described. Examination of the fundus in a patient who is uncooperative or unconscious is not an easy task even for one who is skilled in this work. The patient may need quietening with paraldehyde; the room should be darkened; the pupils should be dilated with homatropine. The aid of an ophthalmologist is advisable.

FIG. 4

The fundus showing petechial haemorrhages and oedematous patches.

Fat in the sputum and urine—Fat has been found in the sputum in patients suffering from fat embolism, but it has also been found in many other conditions and it is not to be regarded as a valuable clinical aid. Fat in the urine is more significant but is not easy to find and is frequently absent. Unfortunately it often does not appear until late in the course of the disease. It is important to remember that fat floats in the bladder on top of the urine and is therefore voided only with the last few drops.

Blood examination and lung puncture—It has been estimated that the smallest embolus which can obstruct a lung capillary has a measurement of 12 microns. It has been recommended that a specimen of blood be taken and a search made for fat globules in excess of this size. Lung puncture is another possible clinical aid and has been carried out in some cases.

Summary of Important Points in Diagnosis

1. History of injury, usually fracture of a long bone or severe soft tissue damage.
2. Interval in which there are no symptoms or signs suggesting complications.
3. Mental changes such as failure to co-operate, restlessness, delirium, stupor, or deep coma. Failure to recover consciousness from an anaesthetic.
4. Alternate elevation and depression of the conscious state.
5. Temperature 102 degrees or more. Blood-pressure normal or raised.
6. Stertorous breathing, cyanosis, frothing at the mouth, moist sounds in the chest.
7. Jacksonian fits and muscle rigidity.
8. Petechial haemorrhages especially at the base of the neck and in the conjunctival sac.
9. White patches of oedema or petechiae in the fundus.
10. Blood urea usually normal. No anuria.

Some of the conditions for which fat embolism may be mistaken are:

1. Secondary shock.
2. Cerebral concussion.
3. Cerebral compression.
4. Blast injury.
5. Crush syndrome.
6. Traumatic uraemia.
7. Badly administered anaesthetic; idiosyncracy to anaesthetic.

TREATMENT

There is no specific treatment and, when the condition has become established, very little can be done. Preventive treatment includes protection from long journeys over rough roads, and care to avoid unnecessary manipulation. Correct splintage is important. Siegmund (1918) showed that the longer the distance of transport to hospital, and the rougher the road, the greater was the incidence of fat embolism.

Deep incision into the wound with evacuation of the haematoma has been carried out by some surgeons on the assumption that local tension increases the liability of fat to enter the venous sinuses. Others, in simple fractures, aspirated the haematoma. Von Klapp (1931) ligated the femoral vein. Various substances have been injected intravenously. Wegelin (1923) injected sodium carbonate in an attempt to saponify the fat. Schanz (1910) injected physiological saline in order to flush out the cerebral capillaries. Yoshimasu and Killian put their faith in adrenalin. Since there is right-sided cardiac dilatation it is reasonable to suppose that intravenous infusion is detrimental. For this reason venesection has been advocated. Oxygen is valuable for cyanosed patients.

Ligation of the profunda vein—In view of the high mortality of fat embolism, and the lack of specific therapy, it has been thought justifiable to ligate the vein draining the injured area. Von Klapp advocated tying either the femoral or external iliac veins. Ligation of the femoral vein is a formidable procedure, not without risk to the limb; and ligation of the external iliac vein is of doubtful value in so far as it does not drain the femoral area.

Experience of ligation of the profunda artery, which proved a life-saving measure in cases of persistent secondary haemorrhage from compound fractures of the femur in wounded and debilitated prisoners of war, and which was never followed by vascular complications in the limb, stimulated me to consider ligation of the profunda vein as a means of arresting venous drainage from the area of the femur. It might be argued that such an operation is akin to shutting the stable door after the horse has bolted, but in this case it does seem that more than one horse is in the stable. The vein was ligated in two cases:

Case 1—Patient with severely comminuted fracture of the shaft of the left femur and small perforation of the skin from within. Twelve hours after the anaesthetic for reduction and immobilisation of the

fracture he became delirious. An extensive petechial rash appeared over the arms, neck, and chest Haemorrhages were present in both fundi and there was one patch of retinal oedema. He was taken to the theatre and the left profunda vein ligated through a vertical incision over the line of the femoral vessels from one to five inches below Poupart's ligament. The femoral artery was exposed and retracted medially to locate the profunda artery arising from its postero-lateral aspect. The profunda vein lies on the antero-medial surface of the artery (Fig. 5). The patient was a little delirious after the anaesthetic and then recovered full consciousness. He did not relapse again and the rash slowly faded.

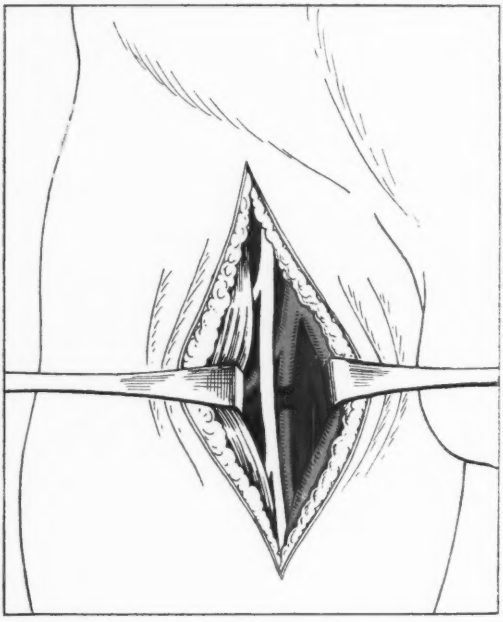

FIG. 5

Operation for ligation of the profunda vein. The femoral artery is retracted medially, distal to the origin of the profunda artery. This exposes the junction of the femoral and profunda veins.

Case 2—The patient had a fracture of the shaft of the right femur and a fracture of the shafts of the left tibia and fibula. He did not regain consciousness after the anaesthetic and remained in deep coma. There were a few petechiae at the base of the neck and in the conjunctiva. It was impossible to tell from which side the emboli arose but, in view of the grave condition of the patient and the success of the previous operation, it was decided to ligate the profunda vein on the side of the fractured femur. The patient, however, remained in deep coma and died four days later.

No conclusions can be drawn from two cases but since the disease carries with it a high mortality, and the operation is simple, it may be considered worthy of further consideration and trial.

SUMMARY

1. Fat embolism occurs in a high percentage of all cases of injury and it is a relatively frequent complication of fractures of the long bones in civilian accidents as well as battle casualties.

2. The diagnosis can usually be established by the clinical features together with certain physical signs which must however be sought deliberately.

3. Important clinical features are the mental disturbance, alternation of coma with full consciousness, petechial haemorrhages in the conjunctiva and skin, and typical changes in the retina.

4. Evidence is still conflicting as to whether the fat arises by embolism from an injured bone, or by general metabolic disturbance.

5. The fat is harmful not so much by reason of mechanical obstruction of vessels as by erosion and rupture of the vessel wall due to the liberation of fatty acids.

6. Preventive treatment appears to be of some value but no satisfactory specific treatment is yet available for the established case.

7. Ligation of the profunda vein has been tried in two patients, one of whom recovered and the other died.

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PLASTIC SPLINTS AND APPLIANCES IN ORTHOPAEDIC SURGERY

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A wide range of plastic materials is now available in industry. It was thought that a number of these might be suitable for the construction of orthopaedic splints and they have been tried with varying degrees of success. Most have been found unsuitable for one reason or another, some because the technique of manipulation was too elaborate, and others because they were lacking in certain necessary qualities. The practical properties required of plastic materials for use in splint-making include: relative ease of manipulation; durability; resistance to fatigue strain; light weight; radiolucency and transparency; inertness to chemical agents; freedom from irritants causing dermatitis; porosity; cheapness. No plastic material satisfies all these requirements but several are available which satisfy most of them. The ideal plastic will be one which after direct application to the patient, or to a mould, will polymerise rapidly to form a rigid material (cf. gypsum) or alternatively one which is soluble in high concentration, about 80 per cent., in a cheap solvent such as water. The curing or setting time must not exceed fifteen to twenty minutes and should preferably be less. Such a plastic when cured or set should have the properties enumerated above. We hope that this goal may be achieved by experimental work now proceeding.

Plastic materials may be divided into two main groups: 1) thermoplastic resins which can be resoftened and remoulded by the application of heat and pressure; 2) thermosetting resins which become more or less permanently hard and insoluble on curing (Worner 1946)[1]. The materials which have been considered experimentally for the construction of splints include: *Thermoplastic*—methyl methacrylate, polyvinyl chloride (P.V.C.), polyvinyl acetate (P.V.A.), P.V.C. & A. copolymer, cellulose acetate, polythene, polystyrene; *Thermosetting—high pressure*—phenolformaldehyde, casein formaldehyde, urea formaldehyde; *Thermosetting—low pressure*—allyl and polyester resins.

It was hoped that new low-pressure thermosetting resins would eliminate some of the difficulties in processing which are found with high-pressure resins, and at the same time have the additional property of high tensile strength which non-laminated thermoplastic materials lack. To date, however, these materials have been found unsuitable for individual splint-making although experimental work in artificial limb production shows promise of success. Of thermoplastic materials, those nearest to our requirements have so far been methyl methacrylate, P.V.C. and P.V.C. & A. copolymer—each having their usefulness in different types of splints.

It should be noted that if great strength is required a laminated plastic is essential. Laminating materials which can be used include glass fibre cloth, cotton, paper, and asbestos. Such lamination does, however, require modification of the usual technique.

External splints using woven cellulose acetate and glass fibre in bandage form have been described (Anderson and Erickson 1945)[2]. The plastic bandage is wetted with a setting solution and applied directly to the patient. A trial of these bandages shows that they have many advantages as compared with plaster-of-Paris bandages, but certain disadvantages have still to be overcome. Their merits include: lightness of weight—not more than one-quarter to one-sixth the weight of plaster of Paris; strength and durability when in complete cast form; coolness in wearing; admission of light and air; capability of sterilisation by autoclaving; the fact that they are waterproof, porous, and unaffected by body secretions; and the ease and cleanliness of application. Nevertheless there are disadvantages. 1) The process involves evaporation of acetone which is an expensive and inflammable solvent.

[1] Australian Plastics, **1**, 26. [2] American Journal of Surgery, **69**, 299.

Great care must be taken that a dangerous concentration of acetone vapour does not accumulate. A case of acute acetone poisoning after the application of casts made of synthetic plaster substitutes in which the solvent-evaporating agent was acetone, and in which the route of absorption was by the lungs, has been reported by Chatterton and Elliott (1946)[3]. 2) Slow setting—As in all evaporating processes a " skin " is formed on both surfaces of the setting splint through which the residual solvent, trapped within, must diffuse before the splint can harden completely. 3) Sharp edges and rough surfaces are left when the cast has dried. 4) Hand-moulding to produce accurate fitting as in the technique of application of plaster of Paris is difficult. The use of crêpe bandage which has been suggested does not give sufficiently accurate moulding.

Acrylic splints have been recommended by Campbell (1944)[4], McGowan (1943, 1945)[5, 6], and Cholmeley (1945)[7]. The obvious advantages are elegance of appearance, low specific gravity, cleanliness in use and radiolucency. Blaine (1945, 1946)[8, 9] holds that these merits are offset by too many drawbacks, but we do not share this opinion. We agree that indirect application by the dental technique is complicated, but there is no need to use such a method. We disagree that " the plastic splint allows sweat to accumulate; pruritis develops and the skin becomes smelly." Ventilation of the splint, as described later, presents no difficulty. Moreover plastic splints do not differ in this respect from plaster-of-Paris splints which are worn continuously for any length of time.

SPECIAL TYPES OF SPLINTS

It might appear that hand manipulation of thermoplastics should be simple, for the reason that the materials become limp and " rubbery " when they are heated. Unfortunately this is far from true. The limp, heat-softened plastic alters shape readily in one or even two planes, but to mould it accurately against a plaster cast of a limb, and to hold it everywhere in apposition while cooling, requires considerable pressure and much skill. It is true that the construction of smaller splints presents relatively little difficulty but for large and complex splints, special technique and apparatus are essential.

Plaster-of-Paris casts are generally used in the manufacture of plastic splints because the temperature to which the plastic must be heated in order to allow successful manipulation is too high for direct moulding to the skin. Attempts to lower the softening point of thermoplastics, so that they can be hand-moulded to the body without burning the skin, can result only in a material which deforms under average living conditions such as sitting in front of a fire or in the sun. So far as practicable only *negative* plaster casts, easily taken by anyone conversant with plaster technique, were used in the production of these plastic splints. This technique is universally used and known. In the making of plastic splints care should be taken, especially where deep curves of small radius are present, to make the plaster cast several inches longer than the splint which is to be designed. Re-entrant curves at the edges of the cast must be avoided. Moreover it is desirable to grease the inside of the cast, or at least its edges and deep folds, with a lubricant such as petroleum jelly in order to allow the sheet plastic to flow easily while being moulded.

Of the two main groups of splints listed below, the smaller type can be made in any hospital with small workshop facilities. The larger type requires experienced technique and elaborate apparatus, but a supply would be practicable under a regional scheme of orthopaedic appliance manufacture without too great a cost of production. *Group* 1—Small splints: 1) limb gutters, 2) angled splints (elbow, knee, ankle), 3) cock-up splints, 4) opponens splints, 5) insoles. *Group* 2—Large splints: 1) anterior and posterior shells, 2) spinal jacket, 3) Minerva jacket and cervical support, 4) hip spica, 5) bucket and pylon, 6) plastic liner.

[3] Journal of the American Medical Association, **130**, 1222.
[4] British Medical Journal, **1**, 152.
[5] Lancet, **1**, 805.
[6] British Plastics, **17**, 30.
[7] British Medical Journal, **2**, 585.
[8] British Plastics, **17**, 331.
[9] Lancet, **2**, 525.

GROUP 1—SMALL SPLINTS

For orthopaedic work, which generally entails the making of individual splints for individual patients, the use of standard shaped plastic splints is inadequate, even though it is claimed that minor local adjustments can be made by simple heating. Anyone who

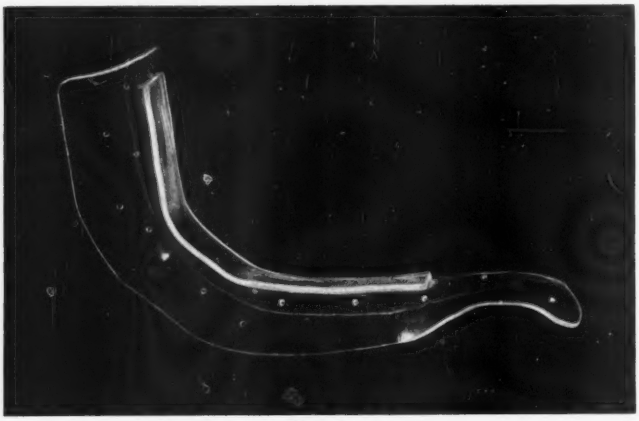

FIG. 1
Angled anterior elbow splint with reinforcing bar.

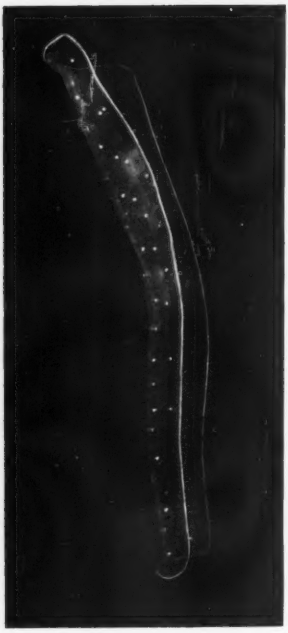

FIG. 2
Knee gutter splint.

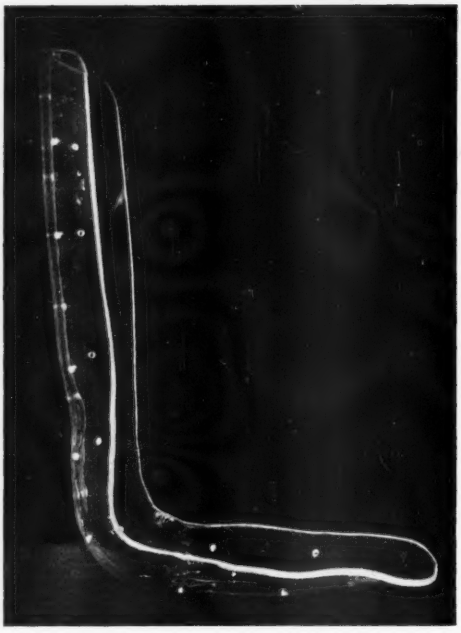

FIG. 3
Ankle splint.

can mould a standard splint accurately to fit an individual patient, can more cheaply make a much better splint tailored to the limb, starting with a negative plaster cast and a length of sheet plastic.

1. Upper and lower limb gutters (one operator, Fig. 2)—These splints are made by hand-pressing heated pieces of plastic sheet of appropriate size and thickness into negative plaster casts (Scales and Herschell 1945)[10]. Such splints can also be made on positive plaster casts. As gutter splints obtained from direct moulding in a negative cast are adequate, it is unnecessary to increase cost and time of production by making positive plaster casts from the negative before processing. When enveloping splints are required, positive casts may be needed.

2a. Angled splints (two operators, Fig. 1)—Anterior elbow and posterior knee splints have been formed entirely by hand from sheet plastic of appropriate thickness (one-eighth or three thirty-seconds inch) cut to the approximate surface area required before

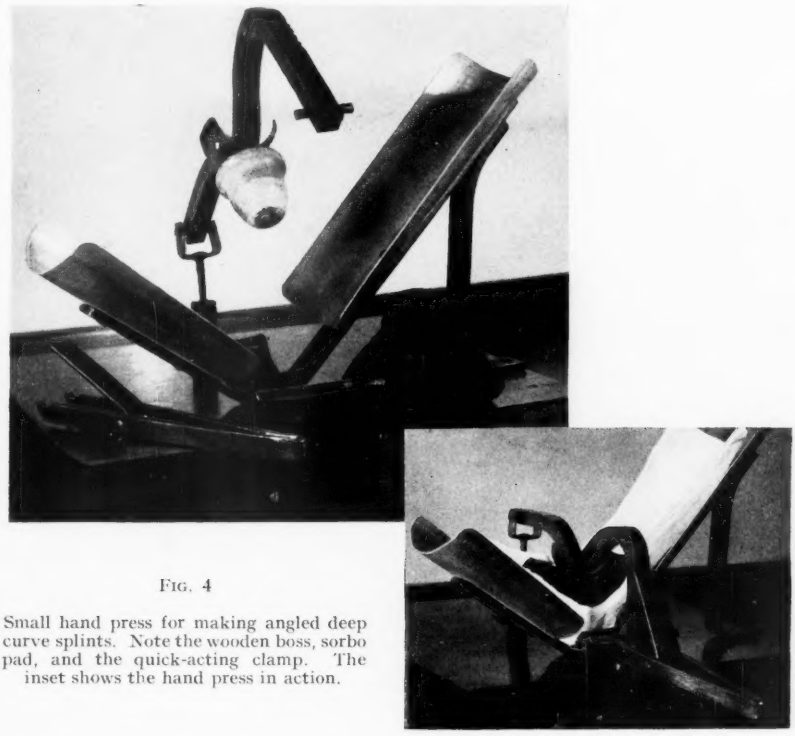

FIG. 4

Small hand press for making angled deep curve splints. Note the wooden boss, sorbo pad, and the quick-acting clamp. The inset shows the hand press in action.

processing. It is important that the width of the strip should not exceed the segmental circumference of the cast at any level because excess material makes accurate hand-moulding extremely difficult. The strip of material is heat-softened and laid in the negative cast by the first operator, who holds it firmly in one half of the splint. The second operator then drags the remainder of the strip over the angle into the other half of the splint, holding it there and moulding it against the inside of the plaster cast until the material has hardened. Since there is considerable leverage on these splints it is advisable to cement a plastic strut across the angle.

2b. Angled splints (one operator)—Posterior elbow and ankle splints (Fig. 3) could not be made in this way because it was found impossible to secure sufficient pressure by hand to

[10] Scales, J. T., and Herschell, W. (1945): British Medical Journal, **2**, 423.

reach the depth required for the heel or the point of the elbow where there are deep narrow curves in two planes. A simple hand press was devised by our technical assistant to overcome this difficulty (Fig. 4). It consists essentially of a right-angled metal rest to carry the plaster cast, bolted to a bench; and a quick-action clamp carrying a boss which is so fitted to the bench that, when it is brought over and locked, it presses a sorbo pad into the heel. The heat-softened tailored plastic strip is laid in the cast, the clamp brought over the heel portion and locked, thus forcing and maintaining the plastic to the full depth of the cast at the heel or olecranon. The foot and calf portion of the splint can then be moulded by hand. Using this simple apparatus the whole operation can be done in a few minutes by one technician.

3. Cock-up splint (one operator, Figs. 6, 7, and 8)—These are made by hand-pressing strips of plastic, which have been cut to approximate size, into a negative plaster cast. When cool, the moulded plastic is trimmed of excess material, and the edge of the palm area is everted to obtain a boss-like effect by careful local heating and finger manipulation.

4. Opponens splint (one operator, Fig. 9)—This splint, originally described by Cholmeley (1945)[7], has been improved and is in common use at the Royal National Orthopaedic Hospital, Stanmore. So far, the best available materials have been found to be P.V.C. & A. copolymer, and methyl methacrylate, the former giving a better " spring " effect. It is made to fit the patient's hand, using only a paper templet. Fig. 10 shows a strip of copolymer, cut to pattern, and the finished splint. The templet can easily be varied in size to fit individual hands. *Technique*: The tailored strip of plastic is carefully softened by heating over a diffused Bunsen flame, and the major curve is set freehand where it is judged that it will turn round the second metacarpal, will allow the tongue to lie in the first interosseous space, and will hold the thumb in the opposed position. The two secondary curves are then formed so as to turn round the estimated positions of the thenar and hypothenar eminences respectively, using careful local heating at the appropriate areas with a very small Bunsen jet flame. The tongue is next locally heated and turned over into the splint, and at the same time the edges of the tongue are fashioned to form two flanges. This flanged tongue will fit over the first web, holding the heads of the first and second metacarpals apart without causing harmful skin pressure. *Note*: All heating of the splint is carried out away from the patient and the splint is applied only when cool. With practice, the number of fittings required during the making of it are few. This splint is the cheapest, easiest made, and most effective type yet evolved for opponens paralysis.

5. Insoles (one operator, Fig. 5)—Valgus insoles with or without metatarsal pads have been made experimentally by our technical assistant, using methyl methacrylate, " hard " P.V.C., and P.V.C. & A. copolymer. The method employed is to press a heat-softened piece of sheet plastic material of the necessary shape and thickness on to a wood last corresponding to the patient's foot size, using a simple hand-press apparatus. The insoles are then trimmed and drilled for ventilation. They have been found very comfortable in use but fatigue strain has occurred in the material, a fault which can probably be eliminated from sheet plastic in the future.

Fixation of small splints—Where a splint has to be held to a limb the methods of fixation include: 1) overall crêpe bandaging; 2) adhesive strapping; 3) slot and strap (Fig. 7); slots are cut near the margin of the splint on both sides at appropriate levels and buckle straps threaded through; 4) stud and strap; shaped studs made from scrap material (Fig. 6a) are pressed into drill-holes in the long axis of the splint, the bases of the studs having been previously heat-softened; when cool these are held securely fixed; encircling soft straps are then fitted, one stud being sufficient for each strap; 5) combined slot, stud, and strap (Figs. 6 and 8). Straps may be either leather or plastic.

[7] Cholmeley, J. A. (1945): British Medical Journal, **2**, 585.

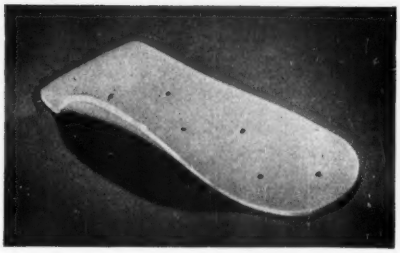

FIG. 5
Insole made of " hard " P.V.C.

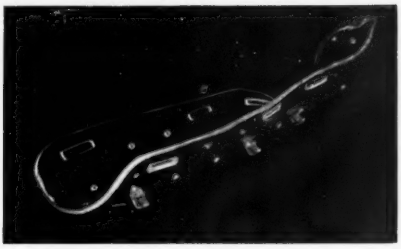

FIG. 6
Cock-up splint with slots and studs.

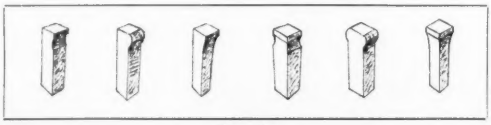

FIG. 6a
Hand-made studs, easily fashioned from scrap material.

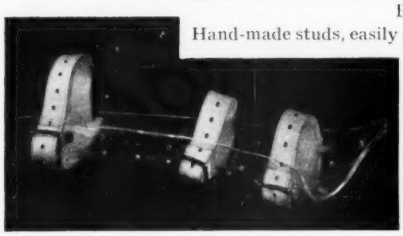

FIG. 7
Cock-up splint with slot and strap fixation.

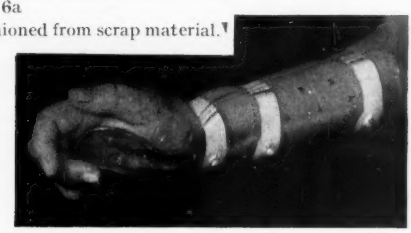

FIG. 8
Slot, stud, and strap fixation.

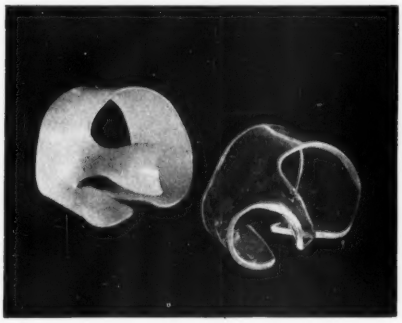

FIG. 9

FIG. 10

Opponens splints. In Fig. 9 the left splint is made of copolymer and the right of methacrylate. Fig. 10 shows a strip of copolymer cut to shape. Note the set of the curves in the finished splint.

Ventilation of splints (small and large)—As already noted, porosity is desirable. Where such plastics as are being used lack this characteristic, ventilation is ensured by multiple small drill-holes of approximately one-eighth inch diameter.

GROUP 2—LARGE SPLINTS

This group requires two operators, a specially equipped workshop, technical skill, and experience of plastics. The splints have been made with thermoplastic material using negative plaster casts and pressure blowing. When made for special cases in which ordinary splinting was inadequate the results have been most gratifying. The stage has not yet been reached,

FIG. 11

Posterior shell mounted on a standard adjustable frame.

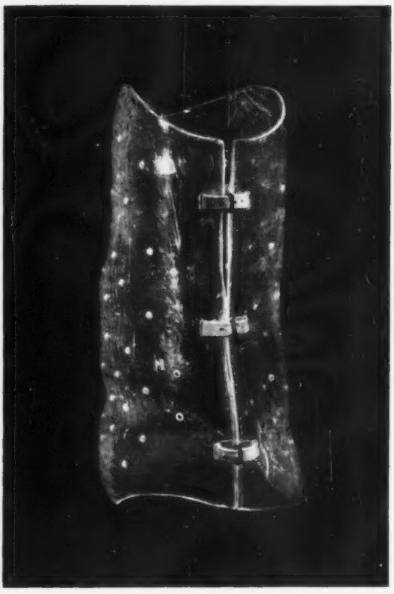

FIG. 12

Spinal jacket.

FIG. 13

Minerva jacket.

however, where the routine use of such splints is practicable, owing to the present relatively high cost of plastic material. Experimental work with plastic laminates using negative or positive plaster casts are promising, and it may be that these can be developed sufficiently for routine use.

When plaster casts are used as negative moulds a modification is necessary to make them suitable for use in the pressure-blowing apparatus. This is a flange which is added by laying plaster slabs across the cast, allowing them to project two or three inches (Fig. 18). In the case of anterior and posterior shells the flanges are formed automatically while the cast is being made.

1. Anterior and posterior shells (Fig. 11)—The method of production of these large body shells has been described previously (Scales and Herschell 1945). It consists essentially in the use of a pressure-blowing apparatus in which a heat-softened sheet of plastic can be blown by compressed air into a negative plaster cast. The plastic mould is then cut out, drilled for ventilation, and mounted on a standard adjustable frame. If required, slots and soft straps can be used for strict immobilisation of the trunk and thighs.

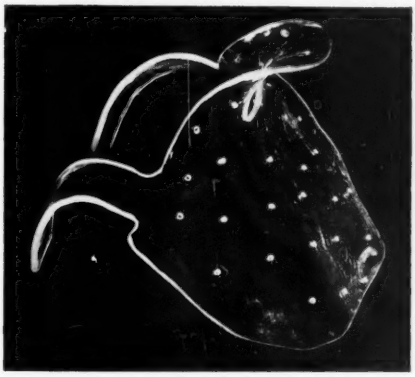

FIG. 14

Cervical support with breast plate, chin support, and shoulder straps—one-piece moulding. No additional fixation is required.

2. Spinal jacket (Fig. 12)—A plaster cast of the trunk is made in the usual manner, and bi-valved along the mid-axillary lines. The two halves are joined end-to-end and the whole is flanged with plaster slabs. When dry, this negative cast is placed in the pressure-blowing apparatus and a softened sheet of plastic material is blown to the shape of the negative mould. After cutting out, two plastic half-jackets are obtained which are finally trimmed, drilled, and fitted. The slot and strap method is used for fixation. The most suitable material for jackets so far used is methyl methacrylate, but it is probable that thermoplastic laminates will supersede it. Unlike celluloid jackets, as made at present with reinforcing duralumin strips, methyl methacrylate jackets need no reinforcement.

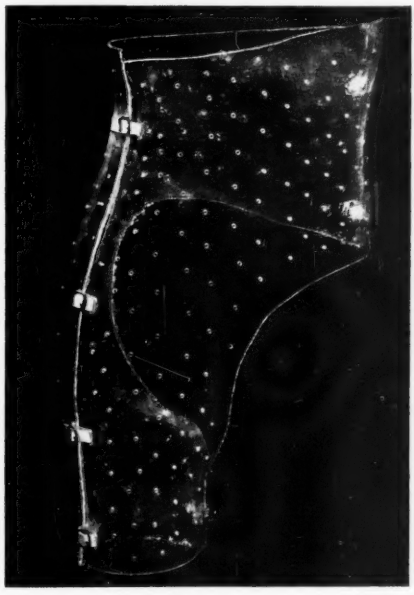

FIG. 15

Hip spica.

A laminate type of jacket has been described by Collinson (1946)[11]. There are, however, certain difficulties in its production which we also have found in experimental work with laminates. The main disadvantage of thermosetting resins such as used by Collinson (1945) is their tendency to cause dermatitis.

3. Minerva jacket and cervical support—These splints were used for special cases where immobilisation of the cervical spine was required. The Minerva jacket (Fig. 13) has

[11] Collinson, H. A. (1946): Lancet, **2**, 215.

been used *in toto* as shown, and also as two separate anterior and posterior splints in cases of laminectomy of the cervical spine. A negative plaster-of-Paris cast is taken, bi-valved, and flanged in the usual way. The two separate halves are used as negative moulds in the pressure-blowing apparatus to obtain corresponding plastic moulds which, after being cut out, form the completed splint. The two halves are held together by buckle-straps threaded through marginal slots. Owing to the depth of blowing necessary to form the forehead portion, the plastic is rather thinned out here and a flat reinforcing band is subsequently attached to strengthen it.

The cervical support (Fig. 14) is made from the anterior half of a negative plaster-of-Paris cast. After pressure blowing, the plastic replica is trimmed so as to leave a breast plate, chin support, and two shoulder straps. An aperture is cut to allow free movement of the thyroid cartilage during deglutition. To permit comfortable rotation of the head, the edges of the chin support are everted or bevelled. This cervical support remains in position without any additional fixation straps and is designed only to prevent flexion of the cervical spine.

4. Hip spica (Fig. 15)—This splint is also made from a bi-valved and flanged negative plaster-of-Paris cast, using the pressure-blowing apparatus. The two halves of the completed plastic splint are held together by buckle-straps threaded through marginal slots.

5. Bucket and pylon (temporary walking prosthesis) (Fig. 16)—Because of the disadvantages of a temporary plaster-of-Paris bucket and pylon, the possibilities of producing a more efficient temporary limb at a reasonably low cost, and without undue delay, were investigated. The disadvantages of a temporary above-knee plaster-of-Paris prosthesis are: *a*) the form of the plaster bucket is such that the weight of the patient is not often taken on

FIG. 16

FIG. 17

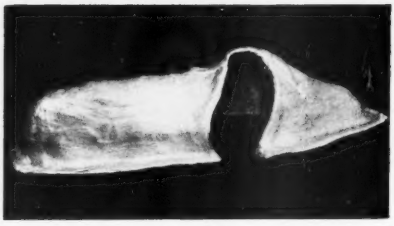

FIG. 18

Bucket and pylon (Fig. 16). Note the simple slot and strap attachment and rubber heel. Fig. 17 shows a plaster cast of an above-knee stump with everted brim for ischial-bearing. Fig. 18 shows the two halves of the divided plaster cast being bridged and flanged; the negative has been left open on one side to show how the two halves are joined.

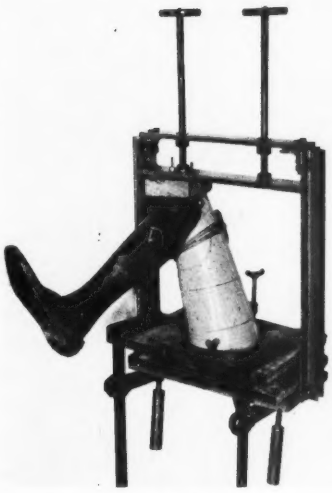

FIG. 19

Simple apparatus for making plastic liner—the limb is held tightly clamped.

the ischial tuberosity as in a permanent artificial limb, but around the circumference of the stump itself; this causes a "step" or "roll" in the natural taper of the stump, and traction on the scar; *b*) for safe weight-bearing the plaster-of-Paris bucket has to be made thick, and when the pylon is attached the whole prosthesis is far too heavy; *c*) the stump sometimes becomes irritated by poor ventilation of the bucket.

Method of production of a plastic bucket—1) *Taking the cast*—The first step is to attach with adhesive tape a length of medium bore pressure rubber tubing obliquely round the upper part of the thigh at the level of the ischial tuberosity postero-medially, and the base of the greater trochanter laterally. This ensures a properly shaped everted brim and ischial-bearing point in the finished plastic bucket. A length of stockinette is then applied over the stump, and plaster-of-Paris bandages used to make the cast. When set the cast is removed, the stockinette drawn out and the cast reapplied to make sure that it is an accurate fit, that it allows full flexion of the thigh, and that it does not press into the perineum (Fig. 17). 2) *Preparing the plaster-of-Paris mould*—When dry the negative cast is carefully divided longitudinally into equal halves (a surface gauge is a great help). These are placed on a flat surface, cut margins downwards, and wide ends facing each other about two to three inches apart. Then by the addition of plaster-of-Paris slabs bridging the gap and projecting all round, a continuous negative mould with flanges is obtained. (See Fig. 18—The cast has been left open one side to show the build-up.) 3) *Making the plastic mould*—When dry, the negative plaster-of-Paris cast is placed in the pressure-blowing apparatus. A three-sixteenth inch sheet of methyl methacrylate of appropriate size is heat-softened, clamped in the apparatus, and blown into the cast. When cool the plastic form is marked out in situ, then removed and carefully cut to yield the two halves. When cemented together with plastic dough these form an exact replica of the original negative plaster cast. The whole process can be completed in forty-eight to seventy-two hours, most of the time being needed for drying of the plaster-of-Paris cast and polymerisation of the plastic dough used

FIG. 20

Finished plastic liner and metal bucket for which it was made. Inset shows the standard "shape" used for making plastic liners.

in joining the two halves together. Ventilation is ensured by drilling the material with one-eighth inch holes after the cementing operation. *Fixation*: The bucket is secured with plastic dough to a plastic pylon, preformed round a mandrel, and the suspension strap passed through slots cut in the cup of the pylon. The prosthesis shown in Fig. 16 has been worn successfully by a patient before the fitting of a permanent artificial limb. It was made entirely of methyl methacrylate, its total weight being 3½ lb. It is suggested that this method of bucket construction could be used in conjunction with a cheap, standard, jointed, and adjustable, artificial limb before the permanent limb is supplied to the patient. This temporary apparatus could be used for more than one patient by the simple process of changing the bucket. In this way the stiff-legged swinging gait which may develop with a straight-knee walking apparatus can be avoided.

6. Plastic liner—When a permanent artificial limb is supplied to a patient it is an advantage to be able to take up the steady shrinkage of the stump which often occurs over prolonged periods. For this purpose a plastic liner was evolved which is an exact fit in the above-knee metal bucket of the limb. It is capable of withdrawal and replacement by another liner of different thickness if required and is individually produced to fit each metal limb without any damage to it. *Method of production*—Standard " shapes " of methyl methacrylate have been constructed in four or five sizes (Fig. 20 inset). In appearance they are rather like a Welsh hat with crown set at an angle to the brim. A " shape " is heat-softened and placed inside the previously warmed bucket of the fully assembled limb. With the knee flexed at 90 degrees the upper part of the limb is held in the apparatus shown in Fig. 19. By clamping the flange of the " shape " between the two base-plates and releasing compressed air into it, the whole shape takes up the exact form of the inside of the bucket. Pressure is maintained until the material has cooled. The shape is removed and trimmed of excess material (Fig. 20). It has been found advantageous to grease the inside of the metal bucket to allow the plastic material to slide easily in it, and so prevent damage to its cellulose finish. Ventilation holes are easily drilled in the liner to correspond with those in the bucket. The time taken to produce such liners is approximately two hours. The apparatus required is not costly or elaborate. The patient is able to continue wearing his artificial limb to which he has become accustomed.

CONCLUSIONS

The advantages of plastics are well known, but there are still some who maintain that these materials are costly and difficult to manipulate. It is not usually remembered, however, that plastics already have their place in orthopaedic splint manufacture, for example in celluloid appliances of many kinds which are in everyday use. They are much lighter than plaster of Paris; they are unaffected by water and body secretions; and some are radiolucent. With the rapid development of modern plastics now taking place there is a wide field for research into their application in orthopaedic surgery. In describing a range of plastic splints and appliances, and outlining the details of their construction, we have tried to show that such research is worth while.

We wish to thank the Royal National Orthopaedic Hospital for facilities given us in carrying out this work, and the Resident Surgeon, Mr J. A. Cholmeley, F.R.C.S., for his interest and help.

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TRAUMATIC URAEMIA

A COLLECTIVE REVIEW

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Traumatic uraemia has been considered largely in association with crushing injuries, but more recent evidence indicates that renal failure with characteristic biochemical changes and clinical features may be due also to trauma of non-specific type [53, 54, 100, 125], and to many other conditions of apparently different etiology [71, 111]. The pathological changes are now well established [37, 100].

HISTORICAL REVIEW

Early references to traumatic uraemia were all associated with injury of a crushing type. Colmers [45], who was sent to the relief of victims of the Messina earthquake, noted that crushing of limbs by falling masonry was almost universally fatal. The condition was largely overlooked during the First World War, despite the crushing injuries of trench warfare [32, 72, 84, 94]. Minami [119] suggested that renal failure was due to the toxicity of myohaemoglobin released from damaged muscles. He based his conclusions on three factors: 1) that the crushed muscles were pale and oedematous, thus suggesting loss of pigment; 2) that there were haemoglobin casts in the damaged kidney tubules; 3) that there was similarity between this form of uraemia and equine paralytic myohaemoglobinuria, a comparison which was confirmed by Gilmour [75]. Bywaters [33] and others described the classical sequence of events which they termed "crush anuria" [14]. This syndrome became well known during the years 1941–43 and most observers accepted the view that release of myohaemoglobin from damaged muscles was the principal cause of the renal failure [21, 36, 39, 68, 101, 112, 115, 117], this view being strengthened by identification of the pigment in the kidney tubules and in the urine [34, 35]. The histological findings were, however, indistinguishable from those of incompatible blood transfusion (the use of stored blood being at that time in its infancy). Nevertheless a case reported by Mayon White and Solandt [116] proved that blood transfusion was not necessarily an etiological factor. Moreover doubt was expressed as to whether muscle damage was the essential cause; and when Glen [76] recorded a fatal case of uraemia after damage to the iliac artery, but without crushing injury, the problem assumed new complexity. Shortly afterwards other writers reported cases with the same histological pattern due to different etiological factors [31, 155, 156], and Foy [71] made the first real attempt to correlate the many conditions which might be responsible.

In 1944 clinicians who studied cases of blackwater fever [109] and gun-shot wounds [53, 125] found difficulty in accepting the view that haemoglobin and its derivatives were nephrotoxic and suggested the theory, supported by experimental evidence [46, 48, 98, 118], that the mechanism of renal failure might be lack of blood supply due to shock, hypotension, or renal vaso-constriction. Maegraith, Havard, and Parsons [111] suggested that the essential etiological factor was "renal anoxia." Nevertheless traumatic uraemia seemed to be associated particularly with crushing injury, and Lucké [100], after examining 538 fatal cases and excluding those due to sulphanilamide poisoning or incompatible blood transfusion, came to the conclusion that in 57 per cent. of cases severe trauma was the important cause.

Despite this clear association of uraemia with injury there has been increasing evidence of similar biochemical and histological changes in the kidneys from many different causes,

TABLE I

Group 1 Shock	Group 2 Electrolyte water balance	Group 3 Pigment	Group 4 Allergy	Group 5 Chemical nephrotoxin
Gun-shot wounds [53, 54, 67, 125]	Haematemesis [18, 15]	Crush anuria [122, 14, 21, 33, 36, 37, 41, 66, 88, 99, 119]	Favism [71]	Mushroom poisoning [100]
Shock [121]	Pyloric stenosis [108], with vomiting [105]	Incompatible blood transfusion [6, 151]	Sulphanilamide (some forms) [16, 22, 134, 132, 154]	Oxalate [63]
Head injury [51, 52]	Acidosis [85, 90, 101]	Burns [9, 114]	Serum sickness [131]	Mercuric chloride [128]
Severe injury [93, 81]	Alkalosis [104, 107, 123]	Yellow fever [111]	Incompatible blood transfusion (some form) [89, 115]	Alloxan
Post-operational azotaemia [15]	Cholera [44, 142]	Hepato-renal syndrone [30, 111]	Pitressin [146]	Phosphorus [128]
Post-partum haemorrhage [155]	Addison disease [43]	Blackwater fever [71, 109, 111, 138, 149]	Epinephrine hydrochloride [146]	The bite of the lactro dictus mactaus (Black Widow) [128]
Concealed accidental haemorrhage [156]	Hypochloraemia [20, 24, 25, 113, 26, 27, 102, 103, 104]	Equine paralytic Myohaemoglobinurea [75]	Staphylococcal toxin [146]	Cresol poisoning [128]
Septic abortion [31]	Dehydration [133, 71]	Weil's disease [111]	Meningococcal toxin [23]	Carbon tetrachloride [152]
Reflex anuria [50]	Diabetic coma [101, 15]	Heat stroke [100]	Typhus fever [3]	Phosphate [61, 107]
Hysterical anuria [146]	Intestinal obstruction [15]	Icterus neonatorum [120]		Uranium [92]
Vascular injury [76]	Repeated haemorrhage [118]			Uric acid [64]
Anoxia [49]	Pernicious anaemia [139, 140]			
CO poisoning [60]	Decreased fluid intake [15, 29]			
	Pneumonia [15]			
	Potassium excess [70]			
	Sodium lack [73, 96]			

and observers have been struck by the similarity between these conditions and both traumatic uraemia and crush anuria. The list of possible etiological factors has now become so vast that they cannot be given in full. They are, however, summarised in five groups in Table I. Despite some overlap, each group is distinguished by an outstanding etiological feature—for example in group 1 the common factor is shock, in group 2 disturbance of the electrolyte water balance, in group 3 presence of pigment in the circulation, in group 4 an allergic basis, and in group 5 factors of chemical nephrotoxins.

Shock and renal function—Ever since the original work in 1937 of Husfeldt and Bjering [93] on renal failure due to injury it has been clear that shock, whether or not accompanied by blood loss, is characterised by failure of renal secretion. Van Slyke and his team [59, 129, 147] showed that when shock was due to bleeding or injury there was progressive decrease in the flow of blood through the kidneys, and that when fluid loss amounted to 40 to 50 millilitres of blood per kilogramme renal blood flow and secretion of water fell almost to zero level. These authors drew analogy between the renal function of dog and man, and stated that in human beings with anuria due to severe and prolonged shock it was possible to restore the kidney circulation by blood transfusion without gaining sufficient renal function to prevent death from uraemia some days later. Other observers noted that after blood transfusion, oxygen extraction of the kidney was not increased proportionately to the increase in other parts of the body, which was even more remarkable in view of the fact that the decreased blood in the kidneys was due to constriction of the renal arterioles. This important observation suggested a possible shunt mechanism whereby certain areas of the renal bed were by-passed. Such a theory is supported by studies in the renal circulation, published in 1947, by Trueta *et al.* [146]. It has been confirmed by other workers who showed that experimental tourniquet shock was accompanied by considerable alteration in the blood flow of the kidneys [95]. Unfortunately it has not been possible to reproduce the conditions of crush anuria in animals [38, 62, 69] because they are unable to withstand the shock of the procedure, and moreover myohaemoglobin in these animals is not in labile form [147].

No less important in determining the essential factor of traumatic uraemia was the work of Cournand [48] and Lauson *et al.* [98] who carried out precise clearance tests in conditions of shock. They demonstrated that there was reduced blood flow to the kidney which might be due in part to renal vaso-constriction but might also be due to changes in renal haemodynamics. This work answered effectively the theory that reduced renal blood flow in shock was due to hypotension (Maitland [112], Tombs [143], and Moon [121], who went so far as to say that the syndrome was a direct sequel of shock). Recently it has been shown that there is neurogenic control of the renal circulation. Trueta [145] observed that over-stimulation of the peripheral nerves in shock might cause persistent spasm of the renal and other arteries. Trueta and Barnes proved that trauma applied to one limb cause widespread arterial spasm extending to vessels of the other limb and to those of the viscera [11]. Trueta and others also showed that renal ischaemia could be produced by stimulation of the splanchnic nerves, and that similar changes could be produced by shock, bleeding, and crush injury. They drew attention to the fact that anuria could be induced reflexly and by emotional disturbance [146].

Electrolyte water balance and kidney function—Disturbance of the electrolyte water balance has been the subject of much research, and it is now established that water deficiency [71, 133], hypochloraemia [20, 103, 113], alkalosis [104, 108, 123], acidosis [85, 90, 101, 96, 73], and loss of available base, are accompanied by rise in blood urea and non-protein nitrogen [15, 29, 113]. These conditions have been ascribed to extra-renal azotaemia for the reason that post-mortem studies have shown no gross histological changes in the kidneys. But evidence in human cases and animal experiments has suggested that there is not only oliguria but also fall in the glomerular filtration rate. Similar changes are found in dehydration due to insufficient fluid intake, intestinal obstruction [15, 29], diarrhoea [44], and

gastro-intestinal haemorrhage [13, 19]. Recent opinions suggest that hypochloraemia is of greater importance than was formerly believed [113] but it is not always possible to determine clinically which of these factors is most essential for the maintenance of renal function because there is a constant effort in the body to maintain and adjust the electrolyte water balance [4]. Kidney function is often restored by correction of the missing factors, but it must be recognised that when renal failure has already supervened, maintenance of the electrolyte water balance requires much judgment and care [5, 54, 110].

Although azotaemia has been thought to result from excessive breakdown of protein it is now suggested that histological changes in the kidney are worthy of more study [15]. There may be degeneration of renal tubules and even cast formation [108]. More recently Verney's work [148] has shown that liberation of an anti-diuretic hormone from the posterior pituitary is dependent upon osmotic pressure in the arterial plasma. It is known that in cases of water depletion, intestinal obstruction, and cholera, there is considerable alteration in the plasma osmotic pressure which may lead to renal failure and azotaemia [113].

Haemoglobin and kidney function—Hitherto most observations have been made in attempting to determine the cause of renal failure in blackwater fever and incompatible blood transfusion. Much of this work is hypothetical, many authors being insufficiently aware of the difficulties of producing a sterile, stroma-free haemoglobin, and often failing to state the amount of haemoglobin used in various experiments. The experiments of Barratt and Yorke [12], Yorke and Naus [153], and Baker and Dodds [10] are misleading. They produced haemoglobinuria in rabbits and found that anuria developed when the animals were acidotic. They concluded that precipitation of haemoglobin in acid urine caused death by obstruction of the tubules and that the remedy was to maintain an alkaline urine. Later investigators, while confirming the significance of acidity of the urine, gave conflicting evidence as to the particular haemoglobin derivative which was responsible [17, 40, 55, 56]. This work has been criticised on the grounds that acidotic rabbits are dehydrated and for this reason are liable to develop uraemia in any case [71]. Gersh [74] injected the haemoglobin of pigs into rabbits and found that cast formation was greater if the animal was dehydrated. Govan and Parkes [80] found that acidosis in the rabbit caused degeneration of the renal epithelium. Convincing proof was also put forward by many workers [8, 13, 28, 57] that haemoglobin and its derivatives were not toxic provided only that they were sterile and stroma-free. This was confirmed by clinical reports, for it was found that haemoglobin is excreted freely in blackwater fever [138], and that uraemia may develop equally in the presence of acidosis, alkalosis, or a normal alkali reserve [109, 149]. These findings are supported by our own observations which showed that severely wounded men often excreted myohaemoglobin without developing uraemia.

Light has been shed on the subject by Yuile *et al.* [158] who showed that when the kidney was damaged by sub-lethal doses of sodium tartate, or by incomplete occlusion of the renal artery, the addition of stroma-free haemoglobin caused fatal uraemia of the type seen in blackwater fever and in traumatic uraemia. Nevertheless the explanation of the initial injury to the kidney is still wanting. Ingenious theories have been postulated. Bayliss [13] suggested that although stroma-free haematin of the blood is non-toxic, globin produces an anaphylactoid reaction which is most marked in the kidney. Later workers suggested that a similar mechanism may be responsible for renal vaso-spasm [89, 115, 127, 150].

Allergy and kidney function—Although it appears that excretion of haemoglobin is not itself the cause of renal failure, it is nevertheless true that fatal uraemia is a common sequel to many forms of incompatible blood transfusion. It is possible that, as a result of haemolysis of blood, an antigen is liberated which not only causes general circulatory failure but has specific vaso-constrictor action upon the renal arterioles, which is then followed by irreversible renal damage [6, 89, 115, 127]. Similarly it has been suggested that sulphanilamide anuria may be due to hypersensitivity of the kidney to combined protein

sulphanilamide, a combination which may occur as the result of intolerance to this group of drugs [16]. In support of this theory, Foy *et al.* [71] believed that the perplexing form of anuria which may follow the eating of broad beans, and is known as "favism," could be explained by protein sensitivity. More recently Black-Schaffer *et al.* [23], studying the Schwartzman phenomenon, produced experimental cortical necrosis of the kidneys in rabbits, presumably the result of an antigen-antibody response to injections of specially prepared cultures of meningococci. Similar results have been secured with staphylococcus toxin [146]. This mechanism may account for the renal changes which are produced by pitressin and epinephrin hydrochloride. The hypothesis is also supported by the finding of azotaemia in serum sickness by Rackemann *et al.* [131].

Chemical nephrotoxin and kidney function—It is well known that certain chemicals have specific nephrotoxic action and that necrosis of the tubular epithelium is caused by such substances as mercuric-chloride, oxalates, phosphates, etc. These substances appear to be precipitated in the loops of Henle as the result of a fall of the pH of the glomerular filtrate [66]*. Such toxic materials may cause necrosis of renal epithelium by direct action, but equally they may give rise to vaso-constriction of the renal arterioles as a reflex defence mechanism, thus explaining reflex anuria [50] and the anuria of incompatible blood transfusion [89, 115, 127].

Such histological findings suggest that in each of the five groups there is sufficient alteration of blood supply to cause necrosis of renal epithelium, but before discussing this statement it is well to examine the evidence from post-mortem and histological findings.

AUTOPSY FINDINGS

Macroscopic findings are few. When death occurs in consequence of crushing injury, incision of the injured muscles shows oedema, swelling, and pallor, with obvious loss of pigment. It is important to note that the skin overlying the injured region may show little or no change. Not infrequently there is also fat embolism with a characteristic petechial rash and changes in the brain, lung, and other organs. There is often oedema of the lung with cardiac dilatation. This appears to result mainly from difficulty in controlling intravenous therapy rather than from the uraemia itself, but it may be due to peripheral vascular failure. Probably the only characteristic change of the syndrome of traumatic uraemia is in the kidney itself which is increased in weight. The capsule is under tension. On section, the cut surface is everted and the cortex is pale; it has a yellow, granular sheen. The pyramids and medulla stand out in contrast and are brick red in colour. When uraemia is due to sulphanilamide therapy, crystals may be seen pouting from the pyramids or in the terminal parts of the tubules.

Histological findings—In tracing the nephron through its course it is found that the glomeruli usually show little or no change, although there is noticeable lack of blood in the tufts themselves. In some areas the glomerular spaces are filled with an albuminous deposit which is most marked round the periphery and is adherent to Bowman's capsule. The epithelium of the first convoluted tubule shows early degenerative change and the cytoplasm stains irregularly; the nuclei are pyknotic and there is usually an eosinophilic catarrh of the lumen. These changes are observed both in traumatic uraemia and in death from intestinal obstruction [15]. The most marked damage occurs, however, in the loops of Henle, for which reason it has been named by Lucké "lower nephron nephrosis" [100]. Degeneration of epithelium in this area is frequently patchy and the tubule is sometimes so distorted as to make it impossible to tell whether it is of the ascending or descending type. The importance of this widespread degeneration is often overlooked because there is a wide

* Since writing this article Harrison *et al.* have shown that this can no longer be accepted, since they have found that the casts removed by micro-dissection are freely soluble in pH of 6·6 or lower, and insoluble in an alkaline medium. (Journal of Experimental Medicine, **1947, 86,** 339.)

variety of casts, mainly of two types, some composed of protein and others of pigment origin. The structure of the casts is of interest; they tend to form round particulate matter such as degenerated epithelial cells or crystals such as sulphanilamide. In many cases the epithelium is stripped and adherent to the outer surfaces of the cast. Another curious cast, which stains red with Mallory and may contain pigment, is granular in appearance and consists of many distinct minute fragments. The ascending convoluted tubule also shows complete destruction with desquamation of epithelium which is stripped in a plicated concertina manner [37]. All types of cast formations are seen. Occasionally the cast is arranged peripherally in the form of a hollow cylinder [42]. The collecting tubules show degenerative changes, often with casts showing evidence of regeneration and polymorphic infiltration. When sulphanilamide has been a factor, the lumen of the collecting tubules may be encrusted with crystals. The intermedullary and interstitial tissue is congested.

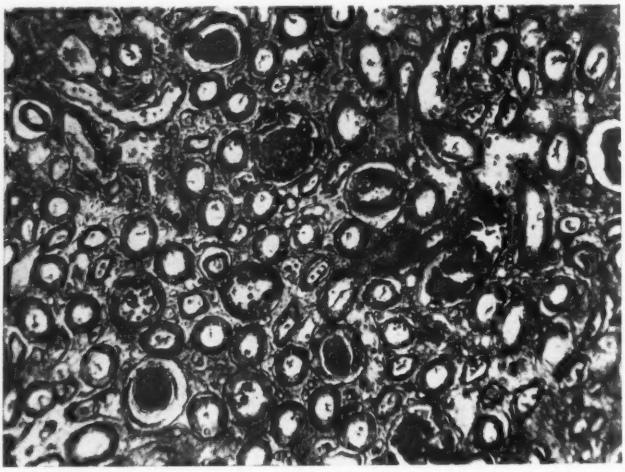

FIG. 1

Section at the level of the loops of Henle showing the wide variety of casts which tends to obscure the tubular degeneration.

One of the most interesting features is the occurrence of venous thrombi in the boundary zone associated with rupture of tubules into the veins [37, 66]. When serial sections have been cut it has sometimes been found that thrombi are adherent to a local lesion of the vein without evidence of tubular rupture [54]. The interstitial tissue at this point is infiltrated with lymphocytes and histiocytes. In other cases this infiltration may be isolated, and when an allergic origin is postulated they may be largely eosinophilic. Other investigators of the renal arterial system have shown the presence of abnormal cells in the juxta-glomeruli complex. Goormaghtich [77, 78, 79] showed that such cells are more plentiful in peripheral cortical areas in the crush syndrome and may also be produced by incomplete ischaemia of the rabbit's kidney. The cells are thought to excrete renin and are said to be responsible for the hypertension which is a clinical feature.

The only other histological finding which may be of significance is necrosis of the central vein of the liver. In patients who survive for several days this is accompanied by reticular replacement and early fibrosis.

DISCUSSION

Of etiological theories which have been advanced, that of tubular blockage has now been generally discarded. Nevertheless Bywaters [41, 42] renewed interest in this theory,

and suggested that oliguria might be due to failure of reabsorption from the second convoluted tubules by reason of the deposition of pigment in the form of a hollow cast. Such a deposit would cause destruction of epithelium and renal failure. A closely allied theory was suggested by Peters [128] who claimed that renal failure was due to rise in the intrarenal pressure. He believed that the primary cause of oliguria or anuria was decrease in the effective filtration pressure caused by interstitial oedema and dilatation of the tubules due to inflammatory changes produced by nephrotoxin. This theory gains support on histological grounds because there is marked interstitial oedema, but the absence of dilatation of the tubules and of the glomerular spaces makes it unlikely.

In a previous communication the author suggested that tubular degeneration was the precursor of this syndrome [54]. It is a matter for debate whether this is caused by nephrotoxic agents or by disturbance of the renal circulation. Supporters of the nephro-

FIG. 2

Section of cortical area showing bloodless glomeruli, degeneration of convoluted tubules, and cast formation in the ascending loop.

toxic theory base their conclusions on the fact that after injury to muscles, pigment may be found in the urine and in the kidney. But the findings summarised earlier in this communication indicate that haemoglobin can no longer be considered toxic unless there is also damage to the kidney. Furthermore the condition is seen also when there is no evidence of haemolysed blood or of intravascular pigment [54, 100, 116]. On the other hand it is possible that the investigations of Green and Stoner [82, 83] may have a bearing, for they demonstrated adenosine triophosphate in increased amounts, both experimentally and clinically, in cases of injury, shock, burns, and ischaemia of muscle. It is not yet clear whether such toxin has direct action upon renal epithelium or whether it produces alteration in the renal circulation.

The possibility that this syndrome may be due to changes in renal haemodynamics has been over-shadowed for some years, for it has been shown already that renal failure is capable of demonstration by more delicate kidney tests in shock and in the majority of conditions classified in Table I.

The recent work of Trueta and others [146] has helped to solve this problem. They have shown that when rabbits are shocked by trauma or bleeding, ischaemia of the renal cortical area develops, with vaso-constriction of the renal arteries, thus suggesting that

there is considerable reduction of blood flow causing anoxia of the kidney epithelium and finally cortical necrosis of the kidney. This theory is supported by histological studies. The fact that renal epithelium is susceptible to anoxia is well known [7, 106, 136, 147]. It is important to know, therefore, the period of time during which anoxia must be maintained before such changes become irreversible. This varies from animal to animal. Allen [1, 2] found that rats were sensitive to renal ischaemia lasting no more than two hours. Van Slyke *et al.* [147] demonstrated that dogs could withstand ischaemia up to five hours and that the duration of renal ischaemia had a direct relationship to the mortality rate. When ischaemia was maintained for no more than three hours all animals were capable of recovery, although there was urea retention for several days; when the arteries were restricted for four hours there was a 50 per cent. mortality rate; and when ischaemia was continued for six hours no animal recovered. On this analogy it is thought that the length of time necessary to produce such changes in the human being may be in the region of eight hours or more. This does not, however, explain the high mortality rate when there is also intravascular deposition of pigment. Yuile *et al.* [158], working with rabbits, produced ischaemia by incomplete occlusion of the renal artery, or by sub-lethal doses of sodium tartrate, and found that when a non-toxic stroma-free haemoglobin was injected there was significant rise in the mortality rate. We confirmed this work and showed that the histological changes of traumatic uraemia could be simulated closely in rabbits by incomplete occlusion of the renal artery except that cast formation was largely of the albuminous type. We showed also that the mortality rate in rabbits after one hour of occlusion was 20 per cent., after one and a half hours 60 per cent., and after two hours 100 per cent. [7]. In subsequent experiments the procedure was repeated and stroma-free haemoglobin was injected intravenously. The mortality rate was not influenced in cases of ischaemia for not more than one hour, but there was significant increase to a mortality of 83 per cent. when ischaemia was maintained for ninety minutes [8]. These experiments suggest that when there is shock sufficient to cause irreversible degeneration of epithelium the chances of recovery are materially affected if haemoglobin is free in the circulation, but that when ischaemia is of lesser degree, and there is sufficient normal renal tissue remaining, it is possible for the animal to excrete haemoglobin without further embarrassment of the kidney.

CLINICAL RECOGNITION

For the traumatic surgeon it is the ability to recognise, prevent, and treat this condition which is important. The earliest sign is anorexia, accompanied by hiccough, and followed within a few days by copious and effortless vomiting. The tongue is dry. The patient is drowsy and he suffers hallucinations with acute attacks of restlessness. Localised areas of oedema may develop. The two most characteristic symptoms, apart from vomiting, are oliguria and hypertension. It may be difficult to recognise the oliguria unless an intake and output chart is kept because these bear direct relationship to each other even although there may be considerable retention of fluid. Examination of the urine may give no indication as to the severity of illness because the specific gravity remains constant between 1014 and 1018. Excretion of urea is fixed between 0·9 mgm. per cent. and 1·6 mgms. per cent. and the pH is held between 5·2 and 6·4. In other cases there may be complete anuria. In the early stages, when shock is severe, the blood pressure may be very low; but as a rule hypertension develops slowly over a period of days, the systolic pressure often exceeding 200 mm. of mercury. One of the most remarkable features of the syndrome is the rapidity and height to which blood urea or non-protein nitrogen may rise. The blood urea is always greater than 100 mgms. and anaemia is common. Other associated changes are rise in the levels of serum potassium and inorganic phosphate, this being accompanied by a fall in the level of serum calcium and serum proteins. There may be reversal of the albumin-globulin ratio.

In a previous paper [5] it has been shown that there is also acidosis and hypochloraemia, but this is not necessarily part of the syndrome since it may also be found in any seriously injured patient. In cases where the limbs have been crushed, amputation does not necessarily prevent uraemia; death usually occurs between the sixth and eighth days [100].

TREATMENT

Preventive treatment—It is estimated that from 16 to 19 per cent. of deaths from gun-shot wounds are due to traumatic uraemia [100], and that when the syndrome is fully developed the mortality rate may be as high as 90 per cent. Preventive treatment is clearly of great importance. Shock must be combated with all possible speed in order to prevent irreversible kidney changes. Before major operations are undertaken the anaemia and electrolyte water balance must be corrected in order that function may be restored in the kidneys before added strain is thrown upon them. Trueta suggested that change in renal haemodynamics may be prevented by splanchnic or spinal block [144]. Porritt *et al.* [130] reported unfavourably on this treatment but success has been achieved by early splanchnic block [86, 87, 135, 137]. When there is limb injury local treatment is important. Patey and Robertson [126] advocated firm bandaging in order to prevent the development of oedema or the release of nephrotoxic substances. Emergency immobilisation is necessary to avoid neurogenic shock.

Correction of electrolyte balance—When once the syndrome is established treatment is much more difficult. Correction of the electrolyte balance may not always be successful but nevertheless it should be put in hand immediately [5, 91, 110]. In assessing the quantity of sodium chloride, sodium bicarbonate, or sodium lactate which should be given the formula of Discombe (1943) has given the most consistent results [58]. The calculation is based upon the approximate weight of the patient in kilogrammes, and the plasma level of chlorides expressed as sodium chloride. The amount of salt which is necessary to correct plasma level is estimated by the formula: $\frac{W(590-X)}{200}$ G, W being the weight of the patient in kilogrammes, X the level of chloride expressed as sodium chloride, and G grammes. The bicarbonate necessary can be calculated by the formula: $\frac{W(64-Y)}{60}$ G, W being the weight of the patient in kilogrammes, Y the level of CO_2 combining power expressed in vols. per cent., and G grammes. In practice this second calculation has not been found necessary unless the level of CO_2 combining power is below 30 vols. per cent. Further disturbance of the electrolyte balance may be shown in the fall of serum calcium and the rise in serum potassium. These changes together may cause serious cardiac complications: if the level of potassium exceeds 40 mgms. per cent. there is danger of fibrillation and death [91]. Sudden death is often observed in cases with oliguria [113]. For this reason care should be taken to avoid sudden and unnecessary movements of the patient. The use of a gastric tube with continuous suction is valuable [91] not only because it avoids the unnecessary distress of vomiting but because it reduces the level of serum potassium, potassium salts being excreted freely in the gastric juices. The calcium level is corrected by intravenous injections of calcium gluconate.

Management of water metabolism—Control of fluid intake is often more important than correction of the electrolyte balance. In consequence of oliguria there is accumulation of fluid within the body with oedema of the lungs and overloading of the circulation. The amounts of fluid needed in twenty-four hours can be estimated by adding the volume of urine and vomit excreted the previous day to the amount of fluid presumed lost by the lungs and sweating (which may usually be taken as 1000 millilitres per day [113]. In order to ensure that the fluid is absorbed it should be given in the form of an intravenous drip. Although this may lead to difficulty in introducing sufficient sodium chloride solution, as much as 4 per cent. can be given without harm provided that the plasma level of sodium chloride does not exceed 475 milligrammes per cent.

Use of sodium sulphate—It has been suggested recently that the administration of sodium sulphate is of benefit [124] but former impressions proved disappointing. It is submitted that sodium sulphate should be used only for the initial stages of sulphanilamide poisoning before kidney tubular damage has occurred. This may cause sudden diuresis with the possible ejection of crystalline deposits of sulphanilamide or haemoglobinuric casts.

Artificial kidney—This new treatment has aroused much interest; it is designed to remove the toxic products resulting from renal failure and to give time for the kidney to regenerate. A sufficient number of cases has now been published to show that the method warrants further study [97]. At the same time it must be recognised that the dialysing fluid is of the simple electrolyte water type which allows passage of water into the circulation of the patient with consequent overloading. Furthermore the machine is bulky. In a paper shortly to be published it is hoped to show how these difficulties have been partly overcome.

Peritoneal dialysis—There are two dangers in the technique of peritoneal dialysis in which large quantities of sterile isotonic fluid are injected into the peritoneal cavity and subsequently extracted. Not only is it difficult or impossible to withdraw all the fluid, but it is difficult to avoid peritonitis due to contamination of the fluid with pathogenic organisms.

Renal decapsulation—Surgeons have advocated bilateral renal decapsulation with the object of relieving increased intrarenal pressure. The procedure is said to be performed easily and often to be followed by diuresis [128]. This was not confirmed by Styron and Leadbetter [141] who performed decapsulation of one kidney and by ureteric catheterisation demonstrated clearly that there was no difference in function of the two kidneys, either in the acute or in the convalescent stages. It would seem that much more justification is needed for the adoption of so heroic a measure in patients who are desperately ill.

SUMMARY

Traumatic uraemia is of particular significance to orthopaedic surgeons in so far as this complication is responsible for high mortality in cases of severe injury, crushing injury, traumatic shock, gun-shot wounds, incompatible blood transfusions, and the misuse of tourniquets. In this contribution the association between muscle injury and renal failure is reviewed.

The syndrome of the "crush kidney," which at first was thought to be the result of deposition of myohaemoglobin in the renal tubules, is almost certainly due to the association of many factors, all of which lead to alteration of the renal circulation and renal ischaemia.

Certainly the combination of ischaemia of the kidney with deposition of pigment and haemolysis of blood causes a high mortality in animals, and it is believed that this may account for the serious prognosis in human cases where there is both shock and haemolysis.

Disturbance of water balance, allergic phenomena, and chemical nephrotoxic action are also discussed. The pathology is considered together with significant changes in the blood chemistry.

A plea is made for early recognition of this clinical syndrome with its characteristic features. Important aspects of treatment are discussed. The dangers of excessive intravenous infusion are emphasised.

Closer investigation and further research promise to throw light on the more accurate localisation of function in the nephron, to add to our knowledge of traumatic arterial spasm, and to explain many orthopaedic problems which hitherto appeared insoluble.

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THE MOVEMENTS OF BONES AND JOINTS

1. Fundamental Principles with Particular Reference to Rotation Movement

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The mechanics of any joint can be divided into two parts: one depends upon the fact that bones are rigid bodies having definite length, breadth, and thickness; the other takes account of the particular arrangement of the musculature, shape of the joint surfaces, and other features which give the joint its individual character. The two parts have often been confused. Effects have been ascribed to muscles or ligaments which in fact depend solely upon the mechanics of dead matter, regardless of the manner in which movements are brought about. Conversely, certain synergic actions of muscles have been unobserved because the circumstances which call for them have been unsuspected. An orthopaedic anatomist will ask how far the movements he studies are of the first type and how far of the second; how far they are brought about by forces under the control of the brain and spinal cord, and how far they are conditioned by the special shape of the articulating parts. Such analysis would constitute a true study of articular mechanics. In this and subsequent articles attention will be directed chiefly to the synovial joints, for these are most frequently of concern to the orthopaedic surgeon.

Movements and displacements—Let us define *movement* as what we observe when examining our patients: *displacement*, then, will be what takes place at the joints which permit these movements. The distinction is not hair-splitting; for the mechanics of movement is rather more simple than the mechanics of displacement and is at first more easily understood.

KINDS OF MOVEMENT

There are two chief kinds of movement: 1) swings, and 2) rotations. Flexion, extension, adduction, and abduction are all swings. The term rotation is to be understood in the sense used in the dissecting room—that is internal (medial) and external (lateral) rotation. In swings, one joint surface slides upon the other; but in rotation one joint surface turns about an axis which is normal or " perpendicular " to the other. No distinction would seem to be so absolute as that between a swing and a rotation. Nevertheless it will be shown that two successive swings are, in general, accompanied by rotation unless synergic action of the muscles intervenes to prevent it. Since this rotation is, as it were, conjoined with successive swings we may call it conjunct rotation. The working of conjunct rotation can be appreciated best by performing a simple experiment upon the living shoulder.

Conjunct rotation at the shoulder—The description of this experiment is adapted from an account in the Irish Journal of Medical Science (MacConaill 1946a).

1) Take an arrow or some object of similar form. Let the right upper limb hang by the side with the head of the arrow pointing forwards and its tail backwards. The arrow lies in a parasagittal plane. The forearm is semi-pronated and is to be kept in this position throughout the experiment (Fig. 1).

2) Swing the upper limb upwards and forwards until it is in a horizontal plane; that is to say bring it into anatomical flexion. The arrow is now pointing vertically upwards.

3) Swing the arm backwards in the horizontal plane until the arrow is in the scapular plane. The arrow is now in the same position as it would have been if the pendent limb had first been abducted in the scapular plane and then rotated laterally through a right angle. This is clearly so because in order to bring it back into its original pendent position, with

the arrow pointing forwards, the limb would not only have to be adducted in the scapular plane but also medially rotated through a right angle.

The result of the experiment may be summarised by saying that flexion of the shoulder followed by backward swing is the equivalent of abduction of the shoulder conjoined with lateral rotation of the free upper limb. By similar manoeuvres it can be shown that abduction of the shoulder followed by forward swing of the arm in the horizontal plane is the equivalent of flexion of the shoulder conjoined with medial rotation of the free upper limb. These facts have been discussed by Fick (1910) and Johnston (1937), the latter being concerned especially with the bearing upon tension of the scapulo-humeral ligaments, but neither writer adventured into the fundamental geometry of the matter. Such study shows that it is a general, rather than an exceptional, phenomenon of articular movements. First, however, let the experiment be continued to its proper end.

FIG. 1

FIG. 2

FIG. 3

FIG. 4

FIG. 5

FIG. 6

Figs. 1–6. Conjunct rotation at the shoulder joint—the first six positions described in the experiment.

4) Having completed stages 1 to 3 of the experiment, adduct the limb to the pendent position without undoing the lateral rotation which it has acquired.

5) Repeat the whole cycle of operations 1 to 3 on the laterally rotated limb.

6) Verify that the cycle can be repeated once only. A stage is reached when full flexion of the shoulder becomes impossible unless the limb is rotated medially *pari passu* with the flexion.

7) Bring the limb back to its original position (*i.e.*, stage 1 above). Rotate it laterally and then attempt flexion of the shoulder. Observe that the amount of flexion which is permitted diminishes with the amount of lateral rotation imposed upon the limb before attempting flexion. Show that this limitation holds also when the limb is subjected to previous medial rotation.

If it is possible to secure a ligamentous preparation of the scapulo-humeral joint, verify upon it that the double cycle of movements (1 to 6 above) brings about a twisting and

tightening of the capsular fibres so that passive flexion of the humerus is brought to a standstill. Show also that previous lateral or medial rotation of the humerus (or converse rotation of the scapula) has a similar effect.

The experiment analysed—We all know that any part of the humerus distal to the shoulder joint moves along a curved line—the arc of swing. The sum of all the possible arcs of swing constitutes a curved surface—the "sphere of movement." This surface is not actually part of a true sphere but there is no loss of generality in considering it to be so.

Fig. 7 is a picture of a sphere. Its "south pole" is shown as well as a portion of its equator (WE) and half of two meridians (ES, WS) which are 90 degrees of longitude apart. Consider the displacement of a small arrow upon the surface of this sphere, To begin with it is placed pointing westwards at the south pole. It is then made to slide along the meridian SW until it reaches the equator. It is then made to slide eastwards along the equator until it reaches the original meridian ES. Look again at the arrow shown at the south pole. Imagine *this* arrow to be slid northwards along the meridian SE. It is clear that the arrow at E will be rotated 90 degrees in a clockwise direction compared with the arrow which has been moved up directly from S. It is also clear from the picture that this rotation has been accomplished during the simple sliding movement from W to E; for the arrow would show no rotation if it were moved back from W to S. This example is a very simple case of the effect of two successive displacements of a rigid body upon a curved surface, and will repay careful study.

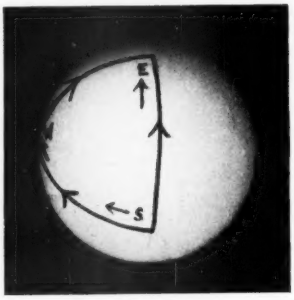

FIG. 7

Diadochal movement is accompanied by rotation of the bone about its long axis.

The arrow can be moved from S to E in two ways: either along the direct path SE; or along a two-leg pathway SW–WE, the two legs having only one point (W) in common. Such a two-leg pathway will be called a diadochal path, and the corresponding movement a diadochal movement. (Gr. *diadochos*, successive.) The arrow-sphere experiment is a special case of the working of a general law, namely that every diadochal movement is accompanied by rotation of the bone about its long axis, unless of course some counter-force is applied to prevent the rotation. Let us call this rotation the conjunct rotation, since it is mechanically conjoined with diadochal movement. We have to ask: what is its nature, and what is its amount?

The answers to these questions are contained in a general theorem which has been stated and proved elsewhere (MacConaill 1946b). This takes account of all possible kinds of surface, and all possible kinds of movement upon them; the "parallelogram of forces" is a minor corollary of it. The most general form of the theorem is needed for an understanding of the working of the diverse types of articular surfaces, but it is not necessary for the purposes of this paper. What is necessary is to consider that part of the theorem which deals with convex surfaces. It can be illustrated in the particular case we are now considering.

First, then, examine the nature of the rotation. Look again at Fig. 1. The diadochal path SW–WE is a clockwise path; the conjunct rotation of our arrow was a clockwise rotation. Had the path been SE–EW, the conjunct rotation would have been anti-clockwise—that is, it would have been of like sense to the sense of the new diadochal path. Expressed as a generalisation: *the conjunct rotation associated with a diadochal movement is clockwise or anti-clockwise according as the diadochal movement is clockwise or anti-clockwise respectively.* Thus, flexion of the right shoulder followed by backward swing of the arm is, from the subject's point of view, a clockwise movement. So also, from his point of view, is lateral rotation of the arm. Again, abduction of the arm at the right shoulder followed by forward swing of the

arm is, from the subject's viewpoint, an anti-clockwise movement. In this case the conjunct rotation is anti-clockwise—that is, medial rotation of the arm.

Secondly, what is the amount of the conjunct rotation? The precise solution of this problem is of theoretical rather than practical interest, but it can be understood easily from Fig. 1. In this figure, the lines SW, WE, and ES cut each other, two by two, at right angles. The whole figure SWE is a spherical triangle, and the sum of its contained angles is three right angles. In this case the conjunct rotation was 90 degrees—that is to say three right angles less two right angles. This result can be generalised: *the magnitude of conjunct rotation is equal to the difference between two right angles and the sum of the angles of the triangle formed by the two " legs " of the diadochal path and the " direct " path between the first and last positions of the bony point considered.* In practice, this magnitude is equal to the angle between the plane of the " direct " swing and the plane of the first stage of the diadochal path. The reader is referred to the paper cited above for the justification of the approximation. It is *not* self-evident.

THE PRACTICAL SIGNIFICANCE OF CONJUNCT ROTATION

The phenomenon of conjunct rotation has a bearing upon three matters of interest to orthopaedic surgeons: 1) the actions of individual muscles and muscle groups; 2) the movements studied in industrial physiology; 3) the diagnosis and therapy of affections of the limbs. Detailed consideration of the first and second topics must be deferred but the general significance of the law of conjunct rotation is clear. The example of the shoulder joint will serve.

When the arm hangs by the side the long axis of the limb—that is to say the axis of medial and lateral rotation—coincides with an axis normal or " perpendicular " to the earth. When the limb is swung first forwards and upwards, and then backwards, the second or backward movement is actually a swing around that vertical axis. We have seen that lateral rotation is thereby produced. In other and more general words: *the result of movement around a standard axis is independent of the particular muscle mechanism by which that movement is brought about.* When the arm hangs by the side lateral rotation is largely the business of teres minor; when it is in a horizontal plane it is largely the business of the back part of the deltoid muscle. The cerebro-cortical " picture " is realised by one set of spinal nerves in one position of the limb, and by another and largely different set of spinal nerves in another position of the limb. The proof of this statement lies in the fact that the teres minor is capable of adding to conjunct rotation by active contraction at a rate exceeding that which it requires for " taking up its slack." The taking up of slack is controlled sub-cortically, whereas the extra *adjunct* contraction is a cortical affair. This concept leads at once to differentiation between two types of synergy, and two types of antagonism.

The simple backward swing of a flexed humerus requires synergic relaxation of the medial rotators if conjunct lateral rotation is to be permitted. There is no consciousness of that lateral rotation, not even in the mind of this writer when he performs it! There is none of that " sense of effort " to which Arthur Lynch has directed attention (Lynch 1923) and which is the accompaniment, however muted, of cortical activity. Furthermore, diadochal lateral rotation of the arm can be carried out passively upon a completely relaxed subject, in whom the cortex is inhibiting rather than relaxing the musculature. The synergic relaxation in conjunct rotation must, therefore, be carried out by a nervous mechanism which differs in part from that which comes into play in adjunct or " active " rotation. Now the " rotator " muscles of descriptive anatomy are deep muscles, and conversely. Hence it may be possible to test spasticity of the deep muscles by carrying out passive diadochal movements and returning the limb to the same plane as that from which it started; inability to maintain the imposed rotation would be a certain sign of spasticity.

Rehabilitation exercises—The phenomenon of conjunct rotation appears to offer a means of exercising muscles which are too weak to raise the limb as they should normally do. Since diadochal movement demonstrates that the action of flexors, extensors, abductors, and adductors includes a rotator component, it follows that rotations carried out actively or passively, in the bed or convalescent chair, will call forth contractions of all the muscle fibres of the joint which is moved. There is no need to enlarge upon this device; it is no doubt widely used already, and all that this writer wishes to show is that such simple exercises have a mechanically rational basis.

SUMMARY

1. Two successive movements at a joint, if not in one and the same plane, constitute a diadochal movement.
2. Diadochal movements impose conjunct rotation upon the bone which has been moved. This may be countered by a rotation of opposite sense.
3. All muscles of a given joint are, therefore, rotators in some degree.
4. Upon the basis of these principles diagnostic and therapeutic suggestions are made.

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POTT'S DISEASE OF THE SPINE WITH RUPTURE OF THE AORTA

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Death as the result of tuberculosis of the spine occurs with sufficient frequency to call for no special comment, but the clinical histories and causes of death in two patients recently treated in this hospital for tuberculous disease of the spine were of considerable interest. In the first case the diagnosis of tuberculous disease was proved, but in the second case their was some doubt.

Case 1. Mrs E. S., 22 years of age, was first seen by the local tuberculosis officer in 1943. Ulcerating tuberculous glands in the neck, previously treated in January 1942 by surgical excision and deep X-ray therapy, had subsequently broken down. Further conservative treatment had been recommended but had been refused by the patient. She was next seen in June 1945 with tuberculosis of the spine involving the tenth and eleventh dorsal vertebrae and the intervening disc. There was collapse of the bodies of both vertebrae

Fig. 1

Case 1. Radiographs, June 1945, showed a large mediastinal abscess pushing the trachea forwards.

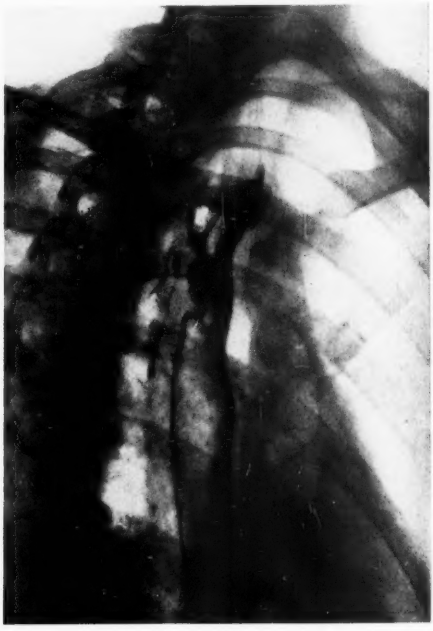

Fig. 2

Case 1. Barium swallow, June 1945, shows leakage from the oesophagus into the mediastinum.

(Fig. 3). Routine X-ray examination of the chest at that time showed a large mass in the superior mediastinum pushing the trachea forwards (Fig. 1). This was thought to be a collection of tuberculous glands which was breaking down, or alternatively a paravertebral abscess arising from a tuberculous lesion in the upper dorsal spine, but there was no radiographic evidence of bone destruction at this level. The abscess ruptured into the oesophagus and caused mediastinitis. At that time the patient was very ill; there was pyrexia with a

temperature of 101°–102° F. She was treated on a plaster bed and her general condition improved. Radiographic examination after a barium swallow showed that the barium leaked out of the oesophagus and tracked downwards in the mediastinum (Fig. 2).

In March 1946 she was admitted to this hospital. Her general condition was poor. She had a hectic flush and evening pyrexia. She was thin and wasted, and her appetite was poor. There were scars of healed sinuses in the right side of the neck and a few small hard glands. No abscess was palpable and there was no paraplegia.

Immobilisation on a plaster bed and open air treatment was continued and although her general condition remained poor, and she developed oedema of the ankles, the spinal disease became quiescent and there was radiographic evidence of sound fusion of the affected

FIG. 3

Radiographs, June 1945, show tuberculosis of the spine involving D 10–11, but there is no evidence of a spinal lesion at the level of the mediastinal abscess or the oesophageal leak.

FIG. 4

Radiographs, November 1946, show that the original lesion at the level of D 10–11 has healed, but there is now destruction and collapse of the eighth dorsal vertebral body.

vertebrae. Repeated X-ray examination of the upper dorsal region failed to show evidence of bone disease and the mass which had previously been noted was no longer evident. In November 1946 an abscess could be felt in the right loin; foul smelling green pus was aspirated and streptococcus viridans was grown on culture of the pus. No tubercle bacilli were found on film examination and no positive result followed inoculation into a guinea-pig. Later radiographic examination showed active disease of the body of the eighth dorsal vertebra with marked destruction and collapse (Fig. 4). The abscess refilled and was aspirated once more but nevertheless a sinus formed. There was marked anaemia (haemoglobin 30 per cent, red cells 1,800,000, colour index 0·85). Three transfusions of blood, one pint each, were given. Diarrhoea was persistent but there was no malaena and no frank blood in the stool.

On December 6, 1946, the patient suddenly felt faint. She passed three pints of blood per rectum. Shortly afterwards she passed another pint of blood. Then she died. There was no vomiting and no blood appeared in the mouth.

Post-mortem examination. At post-mortem examination a little free fluid was found in the peritoneum. There was a large abscess in the right loin extending to the mid-dorsal region. There was no dilatation of large or small intestine, and no blood within the gut, but there was a little staining of one part of the ileum. The stomach contained one and a half pints of blood, the pylorus being tightly contracted. A few tubercles were found on the walls of the small intestine. The spleen was small. The liver and kidneys were normal. In the chest a few pleural adhesions were present but there was no active disease in either lung. The heart was normal.

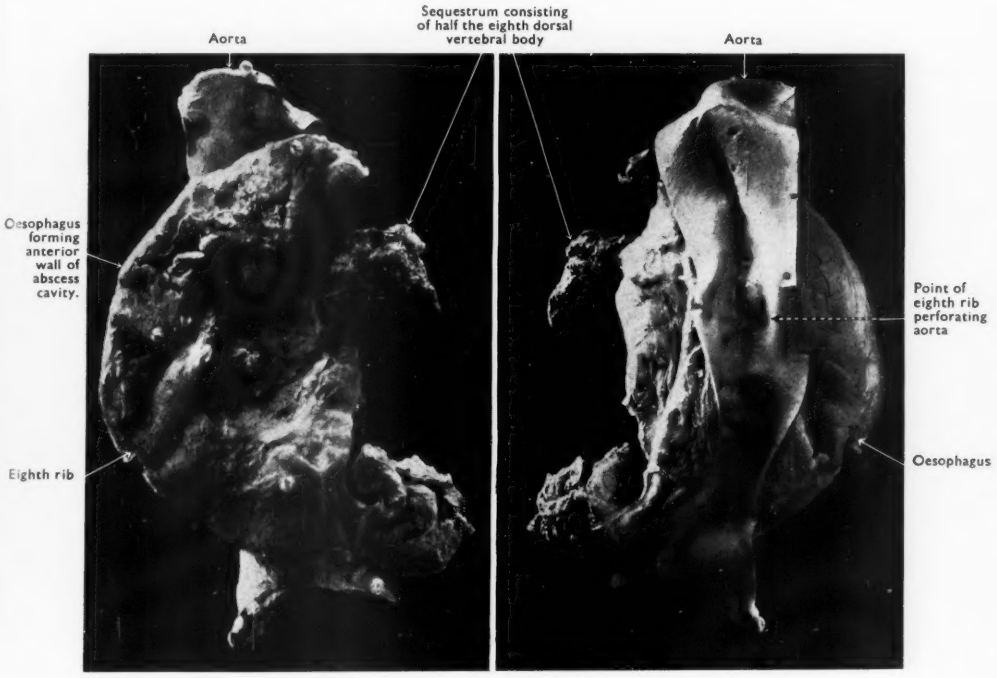

FIG. 5
Posterior View

FIG. 6
Anterior View

Post-mortem specimen showing mediastinal abscess cavity (Fig. 5), the anterior wall of which is the remains of the oesophagus, with the eighth rib sprung forwards in consequence of erosion of its neck, so that it has perforated the oesophagus and the aorta (Fig. 6). The large sequestrum consists of half the eighth dorsal vertebral body; it acted as a ball-valve blocking the upper oesophagus and thus preventing haematemesis when the aorta ruptured.

At the level of the eighth dorsal vertebra, and extending upwards for three inches, was a firm fusiform mass in which the oesophagus and posterior wall of the aorta were involved. There were many surrounding adhesions. This mass proved to be an abscess, the posterior wall of which was formed by carious vertebral bodies and the anterior wall by the remains of the oesophagus, which was almost unrecognisable, and the aorta. Perforating the posterior wall of the aorta was the proximal end of the eighth rib, eroded through its neck by disease, thus producing an aortic-oesophageal fistula (Figs. 5–6). The abscess, measuring three inches long by two inches wide, had a thick fibrous wall. Within it was a free sequestrum, the size of a walnut, consisting of the anterior half of the eighth dorsal vertebra. The whole of this body was destroyed and there was also erosion of the bodies

of the sixth and seventh dorsal vertebrae. The earlier lesion of the tenth and eleventh dorsal vertebrae was healed, but the bony fusion was less sound than had appeared to be the case from radiographic examination.

Comment: This case presents several features of interest.

1. In the first place, early excision of tuberculous glands in the neck appears to have disseminated the disease and accounted for a metastatic focus of tuberculous infection in the tenth and eleventh dorsal vertebrae.
2. Infection of the eighth dorsal vertebra was due to direct spread of infection from the abscess, recognised radiographically in the superior mediastinum, which was originally a tuberculous abscess from mediastinal glands but became secondarily infected with streptococcus viridans from the oesophagus, which at that time appeared intact. No evidence of bone infection in this region was found until eighteen months after the abscess was first observed.
3. The mediastinal abscess subsequently ruptured into the oesophagus, and possibly into the mediastinum as well. Although the appetite of the patient was poor there was no dysphagia; liquid and solid foods were swallowed easily. The post-mortem findings show that the food must have passed actually through the abscess cavity.
4. The next unusual occurrence was erosion of a rib through its neck so that the rib sprang forward and perforated both the wall of the oesophagus and the aorta, thus causing death.
5. Finally the manner of death presents problems for consideration. It would seem reasonable to expect that if a communication was established between oesophagus and aorta, haemorrhage would occur through the mouth. This, however, was not the case; the blood travelled downwards through the intestine and was passed per rectum. Clearly some valvular action had taken place at the upper end of abscess which closed the oesophagus. It is suggested that the large sequestrum lying free in the abscess may have been forced upwards by the influx of blood, thus blocking the oesophagus at its upper end and acting as a ball-valve. It is also interesting that although the stomach was full of blood there was no trace of blood, apart from a little staining in the ileum, from the pylorus to the anus.

Case 2. Mr W. R., aged 37 years, was first seen in June 1944 complaining of pain in the back. Radiographs showed a destructive lesion of the intervertebral joint between the twelfth dorsal and first lumbar vertebrae which was diagnosed as tuberculous (Fig. 7). He was treated in recumbency on a spinal frame for sixteen months and was discharged from hospital in November 1945 wearing a back support.

In April 1946 it was found that the disease had extended (Fig. 8) and antero-posterior radiographs showed a soft tissue shadow which was suggestive of abscess formation. Further in-patient treatment was recommended but this was refused.

In January 1947 he was readmitted and put on a frame. His general condition was fairly good and there was no pain in the back, no kyphosis, and no palpable abscess. Radiographs showed that the area of destruction had increased. Moreover, an extraordinary hollowing of the twelfth dorsal vertebral body had developed (Fig. 9) and the possibility of neoplasm of the spine was suspected. Punch biopsy in October 1947 showed no evidence of tumour. There was pus, with cellular debris, but no micro-organisms, and cultures remained sterile. The Wassermann and Kahn reactions were found to be strongly positive and a course of anti-syphilitic treatment was instituted.

In November 1947 the patient complained of pain in the left loin, of sudden onset, accompanied by nausea, vomiting, and a sensation of fullness in the abdomen. Clinical examination revealed a large, firm mass in the epigastrium extending under the left costal margin with an upper limit which could not be reached. The swelling pulsated but it was difficult to determine whether the pulsation was expansile or transmitted. Radiographs

FIG. 7

June 29, 1944

FIG. 8

April 4, 1946

FIG. 9

August 12, 1946

FIG. 10

December 2, 1947

Case 2. A destructive lesion at the level of the D 12, L 1, was diagnosed as tuberculosis (Fig. 7). During the next ten months the disease extended (Fig. 8). Unusual hollowing of the twelfth dorsal vertebra then developed (Fig. 9). Two months later typical aneurismal erosion was obvious (Fig. 10).

showed still further destruction of the front of the vertebral bodies which by then was characteristic of aneurismal erosion (Fig. 10). The question remained whether the erosion was due to an aneurism of the abdominal aorta, coincident with a tuberculous or gummatous lesion of the spine which happened to be at the same level; or whether vertebral erosion might be due to the transmitted pulsation of a tuberculous spinal abscess. If the latter alternative was correct the abscess was obviously approximating so closely to the abdominal aorta that there was danger of erosion of the wall of the artery with secondary rupture into the abscess cavity such as had occurred in the first case. In this event evacuation of the abscess through a posterior approach was desirable; on the other hand such a procedure was not to be undertaken until the surgeon was sure that he might not open an aneurism! An abdominal laparatomy was therefore carried out and the mass was found to be a large aneurism of the abdominal aorta extending above the diaphragm. The patient made an uneventful recovery from the operation. On January 12, 1947, he complained of sudden dyspnoea and pain in the left side of the chest. He died in twenty minutes.

Post-mortem findings—Post-mortem examination showed that the left pleural cavity was full of blood. The arch of the aorta showed extensive syphilitic aortitis but no aortic incompetence. The heart was normal but there was excessive fluid in the pericardium. A large mass, extending through the diaphragm, proved to be a very large aneurism of the aorta extending from the level of the third lumbar vertebra to that of the seventh dorsal vertebra, lying chiefly in the left side but also extending to the right side of the mid-line below the diaphragm. In the intra-thoracic part there was a perforation rather more than two centimetres in diameter. The wall of the aneurism was very friable indeed. It was not opened. The whole specimen including the vertebral column was removed in one piece and preserved in fixative.

Most unfortunately, two days later the hospital was destroyed by fire. The pathological department and this particular specimen were lost. No dissection has been possible.

Discussion—Owing to loss of the specimen the diagnosis cannot be stated with certainty, and even the correct sequence of events is left in some doubt. The initial radiographs (Fig. 7) suggested that in the first place there was a simple destructive lesion of the spine which was probably tuberculous but might have been gummatous in origin. Later radiographs showed striking increase in the destructive process, with change in its nature from that of a simple intervertebral inflammatory lesion to that of typical aneurismal erosion. It is believed that a syphilitic aneurism of the aorta developed at a later date than the spinal lesion, and because the bone behind it was softened by incompletely healed disease the aneurism caused secondary pressure erosion. It is interesting to note that although the aneurism extended over eight vertebral bodies only those involved in disease showed erosion.

Re-examination of the antero-posterior radiographs taken in September 1946 shows evidence of two soft tissue shadows, one overlying the other; presumably one was due to the spinal abscess and the other to the aneurism. The radiographs taken three months later, and two weeks after the sudden onset of pain in the left loin, show loss of the clear-cut shadow on the left side, a more fusiform shadow on the right, and a new shadow of a large mass in the left chest. It may be that the sudden attack of pain was due to rupture of the aneurism into the abscess cavity, with subsequent upward extension of a false aneurism into the mediastinum which finally ruptured into the left pleura.

Unfortunately there are a number of points which cannot be determined with certainty. 1) Was the bone lesion of the spine tuberculous or syphilitic? 2) Was the aortic aneurism secondary to the spinal lesion or was it coincidental? 3) Did the aneurism rupture into an abscess cavity and into extra-peritoneal tissue causing a false aneurism, or was it a true fusiform aneurism which increased rapidly in size until it ruptured?

We are indebted to Sir Reginald Watson-Jones for permission to report these cases, and to Dr Paton Phillip, Tuberculosis Officer for Cambridgeshire, for early clinical details and radiographs of the first case.

CONGENITAL ABSENCE OF THE HUMERAL HEAD

Report of Two Cases

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The purpose of this article is to present clinical data, review the literature, and speculate as to the origin of a rare condition. The first case here recorded had been passed over with the remark that "the condition is probably congenital." Attention was aroused when a second case was seen a few months later.

CLINICAL MATERIAL

Case 1. B. S., aged 26 years—Of normal appearance, with powerfully developed muscles. Abduction and medial rotation movements of the right shoulder joint were almost completely lacking. The right humerus was ten inches long and the left eleven and a half inches long. There was no history of injury and no other explanation of the condition which had been present as long as the patient could remember.

FIG. 1

Case 1. Congenital absence of humeral head, showing the flat surface of the humerus, the well-developed greater and lesser tuberosities, and the outward bow of the shaft of the humerus.

Radiographs showed that in place of the usual head of the humerus there was a flat surface with a small central boss projecting about 4 mm. (Fig. 1). The articular surface was twice the length of that of the glenoid. The greater and lesser tuberosities were well developed and the inter-tubercular sulcus was seen clearly on the outer surface of the bone. The shaft had a distinct outward bow with its summit at the deltoid insertion. The cortex was abnormally thick and dense on the outer side, but it was thin on the inner concave side. The glenoid was flattened, with a slight concavity opposite the central boss of the humerus. A tubercle was present above the glenoid. The coracoid process was developed normally. The left shoulder joint was normal.

Case 2. Z. M. K., a healthy Sepoy, aged 28 years—This patient stated at once that disability of the shoulder joints had been present since childhood but not to such a degree as to affect his capacity as a fighting man. He was somewhat indignant that his fitness for service should be questioned. On examination he was found to be normal except for the upper arms and shoulder joints. He was well developed. Both upper arms were short but the forearms were of normal length (Fig. 2). The right humerus measured nine inches and the left ten and a quarter inches. Judging by the position of the lateral condyles there was lateral rotation deformity of the right humerus. Abduction was limited by bone block (Fig. 3). *Radiographs* showed gross deformity of both humeral heads and glenoids. As in the first case, the humeri were shortened and bowed outwards and the cortex was thicker on the convexity and thinner on the concavity. In order to gain information as to the shape of the joint surfaces radiographs were taken in the position of adduction, abduction and lateral rotation, and also from the axillary view. The findings were:

FIG. 2

FIG. 3

FIG. 4

FIG. 5

Case 2. Bilateral congenital absence of the heads of the humerus. Note the shortening of the upper arms (Fig. 2). Abduction movement of the shoulder is limited by bone block (Fig. 3). In both arms the shaft of the humerus is bowed, there is alteration of cortical density, and the glenoid boss is a convex protrusion fitting into a concave humeral head (Figs. 4 and 5).

Right shoulder—The glenoid was represented by a boss of bone at the upper end of the axillary border of the scapula. Above and below this rudiment, separated from it by well-defined grooves, could be seen the supraglenoid and infraglenoid tubercles; these structures and the coracoid process appeared to be of normal size. The upper end of the humerus was represented by four bone elevations surrounding a concavity, the elevations representing the greater tuberosity, the lesser tuberosity, and the rather exaggerated facets of the infraspinatus and teres minor muscles. The shaft of the humerus showed bowing and alteration of cortical density (Fig. 4). The axillary view showed that the glenoid boss was a convex protrusion fitting into a concave humeral head. *Left shoulder*—The glenoid boss was larger than on the right side, and the humeral joint surface was more shallow and more vertical. Its lower lip was composed of dense bone and was pressed down. The supraglenoid tubercle was not differentiated. The lesser tuberosity was more pronounced than in the right humerus but the greater tuberosity, and the bowing, thickening, and trabeculation of the shaft, were similar (Fig. 5). An axillary view showed similar reversal of the ball and socket arrangement of the joint.

REVIEW OF LITERATURE

Extensive search of the literature has disclosed only six cases with deformity similar to that now described. Lewin (1931) reported two cases. The first had bilateral but asymmetrical deformity. On one side there was a convex glenoid, and a concave humeral head, suggesting absence of only part of the capital epiphysis (Fig. 7). On the other side the joint was formed of a humeral surface which was slightly S-shaped, filling a corresponding glenoid surface (Fig. 6); there was no vestige of the head and the glenoid was more developed than its fellow. Lewin's second case showed unilateral deformity very similar to that of his first case, but it was complicated by ankylosis of the elbow and Madelung's deformity (Fig. 8). Walter Müller (1939) described three cases with deformities of varying degree (Figs. 9, 10, 11). All three patients had other congenital bone and joint deformities. Brailsford (1944) recorded another case. No details were given but it will be seen from the radiographic tracing that the appearances are similar to those of Lewin's first case (Fig. 12).

DISCUSSION

This group of cases represents varying degrees of a typical but hitherto unrecognised deformity of the shoulder joint. The essential features are complete or incomplete absence of the humeral head with a rudimentary glenoid. The deformity may be unilateral or bilateral; it may be isolated or it may be one of a number of deformities in the same patient. It is associated with relatively slight disability. Both patients whose cases are now recorded fought as combatants through the 1945 Burma campaign. The ball and cup arrangement of the shoulder joint is reversed or it is replaced by a saddle-shaped articulation. There seems to be correspondence between the degree of capital deficiency and the state of development of the glenoid (Müller's three cases—Figs. 9, 10, 11). It appears that lack of the capital epiphysis, or of its cartilaginous and precartilaginous stages, causes corresponding lack of the upper epiphysis of the glenoid-complex while the main body of the glenoid continues to develop.

These observations suggest that the defect occurs at a very early stage of embryonic life and that it is due to failure of development of the tissue in which the capital epiphysis should form. The observation that centres developing late and outside the joint capsule are present and well formed, while those developing early and inside the capsule are not present, or are present only in rudimentary form, also indicates that the defect occurs in the early period of development of the primitive joint.

Further support for this hypothesis is gained from consideration of the experimental work of Murray, Fell, and Robison. Murray (1926) showed that joints develop characteristic shape even when grafted elsewhere so that they are remote from the normal influence of contiguous structures. Clearly the general form of a joint is determined by intrinsic influences, although structural details may be the result of "mechanical" forces. Fell and Robison (1929) came to similar conclusions. The problem as to how two opposing rudiments, developed

from a common mesodermal mass, are able to separate and become movable and yet be interdependent and complementary, was considered by Fell and Canti (1934) using Canti's technique of cinematography in observing and recording *in vitro* the development of tissues (1928). Fell and Canti showed that joint formation is not caused by the presence of a layer of non-chondrogenic tissue across the site of the future articulation as suggested by the studies of Murray (1926) and Warren (1934). These studies demonstrated that the avian limb bud is a mosaic system and suggested that separation of the articular surfaces might be an expression of this already existing mosaic. Fell and Canti, in the same report, produced

FIG. 6
Lewin's first case—right.

FIG. 7
Lewin's first case—left.

FIG. 8
Lewin's second case.

FIG. 9
Müller's first case.

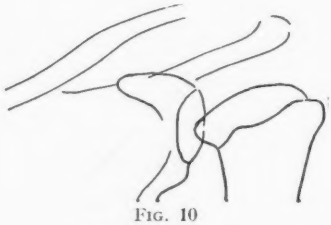

FIG. 10
Müller's third case.

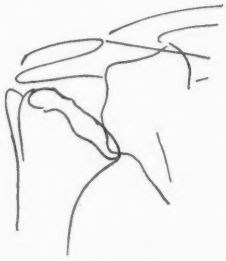

FIG. 11
Müller's second case.

FIG. 12
Brailsford's case.

evidence for important conclusions. 1) The earlier stages of joint formation can proceed in the complete absence of muscular action and of nerve and blood supply. 2) Joint formation can take place in the absence of the presumptive joint region. 3) Joint formation in the absence of the presumptive joint tissue is controlled by two factors—(*a*) the amount of tissue removed, (*b*) the degree of development of the blastema up to the time of operation. 4) The articular ends of the long bones cannot develop their normal shape after removal of the presumptive articular tissue.

From these considerations it may be supposed that the deformity under consideration arose at the presumptive stage, since only those parts entering into the formation of the primitive joint appear to be affected. Extra-capsular structures developed normally except

for minor variations which may be ascribed to altered mechanics. At a very early stage, when the primitive joint had just begun to form, an accident occurred which altered the process of separation in the mesenchyme to form the articular ends. The brunt of force of the accident seems to have fallen upon the humeral head, because in all these cases the glenoid is at least present in rudimentary form, whereas the complementary head of the humerus is seriously deficient in every case. When the cases are set out in order it is seen that the degree of development of the glenoid bears inverse relationship to the extent of humeral defect. If the humeral defect is minimal, the glenoid is well formed; if the humeral element is missing completely, the glenoid is a mere rudiment.

The presumptive joint stage appears to have been well advanced when this unknown factor exerted its influence, causing virtual removal of the caput area and leaving the glenoid without possibility of developing its normal shape. If this is a true reading of the picture, these cases present clinical evidence in support of the conclusions reached experimentally by Fell and Canti.

That the damage is inflicted on the shoulder alone, or only a few joints, might suggest that the accidental factor was present for a limited time which was coincident with formation of the presumptive shoulder joint. But it would appear also that some parts of the body may be more sensitive than others even when exposed simultaneously, and this would account for unilateral cases.

It is possible that the cases collected here on the basis of similar radiographic appearances in the shoulder may not form a true group, because they may not have a common cause. The clinical evidence of similar appearances is, however, supported strongly by experimental data, and it seems reasonable to accept these cases as a group which show a congenital deformity of the shoulder joint, hitherto unrecognised.

SUMMARY

1. Two cases of an unrecognised congenital defect of the humeral head are described and the cause is discussed.

2. Only six cases with similar radiographic appearances could be collected from the literature. In most of these cases other skeletal deformities were present, whereas in those now reported only the shoulder was affected.

3. Examination of radiographs suggests that the main deformity consists of lack of development of the capital epiphysis of the humerus.

4. Consideration of the cases, together with experimental data from the studies of Fell and Canti, suggests that the time in development at which the fault occurred was the presumptive joint stage, just when the articular rudiments had separated.

5. A " nociferous agent," acting only for a limited period, and only on certain tissues, is postulated.

6. It is suggested that the defects recorded should be recognised as a group of congenital deformities of the shoulder joint.

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NAIL IN THE SKULL

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A male Chinese, aged thirty years, was on his way to Court to answer a charge of attempted suicide. He sat on the roadside. He took a large nail in one hand and a brick in the other. He hammered the nail into his head. No symptoms developed and he went on his way. But the next day, because a large nail was still projecting from his skull, he was admitted to the Singapore General Hospital. Radiographic examination showed that the nail had been driven accurately through the mid-line (Fig. 1). A surgeon decided that although there were still no symptoms the nail should be removed. Immediately he did so there was profuse haemorrhage from the longitudinal sinus and the patient died.

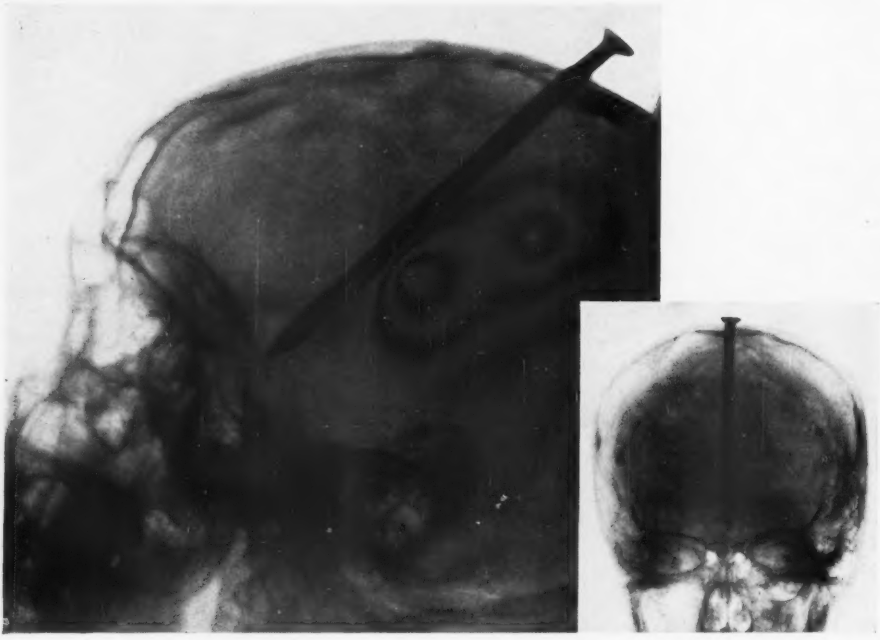

Fig. 1

A male Chinese drove a nail into his own skull with a brick.

I have radiographs of a second case in which a rather smaller nail had been driven into the mid-line of the skull but unfortunately no clinical history is available.

Another case is recorded: "Sisera lighted down off his chariot and fled away on his feet. . . . And Jael went out to meet Sisera and said unto him, 'Turn in, my lord, turn in to me; fear not.' And when he had turned in unto her into the tent, she covered him with a mantle. . . . Then Jael, Heber's wife, took a nail of the tent and took an hammer in her hand, and went softly unto him and smote the nail into his temples and fastened it into the ground; for he was fast asleep and weary. So he died."

FROM AN ATLAS OF GENERAL AFFECTIONS OF THE SKELETON

2. OSTEOPETROSIS

Osteopetrosis generalisata, Marble bones Albers-Schönberg's disease, Osteosclerosis fragilitas generalisata

H. A. THOMAS FAIRBANK, LONDON, ENGLAND

The condition described by Albers-Schönberg in 1904, and to which Karshner (1926) gave the name of osteopetrosis, is a rare developmental error, the chief characteristic of which is excessive radiographic density of most or all the bones of the skeleton.

Familial and hereditary tendencies—A distinct familial tendency is displayed and the disease is occasionally inherited; for example, eight cases occurred in three generations (McPeak 1936). It may be transmitted by either sex. Any special features which may be present are constant for a particular family. Consanguinity of parents has been noted; Nussey (1938) reported consanguinity in twenty-three of 112 cases.

Sex—The disease affects both sexes, males slightly more frequently than females.

Age—It has been found at all ages from foetal life to seventy-five years. It undoubtedly begins before birth in some cases, and possibly in most.

Etiology—The cause is unknown. Rather surprisingly, some have believed that parathyroid hyperactivity is responsible (Dupont 1930, Ellis 1934), A tumour of the parathyroid was present in one case but this was regarded, probably correctly, as secondary and defensive (Péhu *et al.* 1931).

Clinical features—The chief features of the syndrome are abnormal density of the bones with or without fragility, a strong tendency to anaemia which may be severe and fatal, and optic atrophy. The severity and course of the condition vary considerably. It may run a benign or a malignant course; according to Nussey (1938) benign when the condition is inherited, and malignant when there is consanguinity of the parents. In a well-marked case, assuming that the patient survives, the formation of abnormally dense bone continues till growth ceases, the condition of the skeleton then remaining stationary, at any rate radiographically, though it may progress later. There may be intermissions, remissions, or complete cessation of the developmental error before growth ceases, all of which are demonstrated clearly in X-ray films.

Tendency to fractures—As a rule the bones are intensely hard like marble, but bones much softer and more like chalk were present in at least one case (Pirie 1930). A tendency to fracture is certainly present in some cases. One patient sustained ten fractures by the age of twenty years. On the whole fragility of the bones has been much exaggerated. When they occur fractures are sharp, abrupt, and transverse. Callus formation may be slow.

Anaemia—Usually there are no symptoms except those resulting from anaemia, which may become severe enough to call for investigation and may even be fatal at any age. It results from reduction of the blood-forming marrow in the sclerosed bones and eventually becomes of the aplastic type. Cases in which it was leukaemic and even apparently pernicious in type have been described. The anaemia is a source of real danger but it by no means corresponds always to the severity of the bone condition. A woman lived to the age of seventy-five years in spite of advanced petrosis of the whole skeleton and yet section of a femur in this case showed complete obliteration of the medullary cavity. Apart from the anaemia, blood examination reveals nothing of importance. The serum calcium is usually normal, but it is raised occasionally. When generalised calcinosis is present the serum calcium may even be doubled (Schulze 1921). The plasma phosphatase is either normal or slightly low. Children displaying

widespread abnormal deposits of calcium salts in the body may or may not have osteopetrosis in addition. If anaemia is severe, compensatory enlargement of the spleen, liver, and lymph glands may be present.

Optic atrophy—Optic atrophy may result from changes in the base of the skull but it is frequently absent. It has been seen as early as the sixth month of life. Nystagmus, hydrocephalus, facial, or ocular palsy, and deafness are other not very uncommon complications. The teeth are prone to decay. Some degree of dwarfism is often present. Osteomyelitis is stated to be not uncommon.

Radiographic features—As seen in radiographs, the extent to which bones are affected varies considerably. The whole of a bone including the epiphyses may be uniformly dense and completely structureless. With rays of sufficient hardness fine striations, both transverse and longitudinal, may be made out in the metaphyses and epiphyses (Sear 1927). Occasionally less dense areas may be seen in the sclerosed bone. The parts unaffected by petrosis may show osteoporosis but this is sometimes more apparent than real. As a rule metaphyses are more affected than shafts of the long bones; they are commonly enlarged or clubbed, the enlargement ending abruptly at its junction with the diaphysis. Occasionally the reverse holds good, the diaphyses being more affected, with the metaphyses less dense and either normal in shape or clubbed (Ellis 1934). A streaky or patchy arrangement of the denser bone is very rarely seen in cases of generalised osteopetrosis. Apart from clubbing, the shape of bones in both varieties is unaffected. In some cases a narrow clear transverse band marks the junction of diaphysis and enlarged metaphysis: in others, dense and less dense bands alternate. The unusual distribution of dense bone may suggest the markings of a zebra. The clavicles, the lower part of the humerus, and the radius and the ulna, may show less density than the rest of the skeleton. If, in a child, the tendency to form petrosed bone ceases suddenly after a time, the epiphyses will show a dense centre surrounded by a halo of bone of normal density. The ilia often show alternating dense and clear curved bands parallel to the crests. The carpal and tarsal bones, though sometimes universally dense or mottled, frequently show a dense centre surrounded by a halo of clearer bone; or the reverse may be seen when the pathological change began later than usual. Alternating concentric rings of dense and clear bone are not uncommon. As a rule the phalanges are decidedly less affected than the rest of the skeleton and they show a dense transverse band of varying width in the metaphysis, close to, or a short distance from, the epiphyseal lines. In milder cases these bands are a striking feature of the hands. Similar dark bands in the phalanges and at the ends of the long bones may, however, result from the absorption and deposition of lead, phosphorus, or bismuth. The skull shows the expected degree of density which is particularly marked at the base. The pituitary fossa is usually rather small, and the posterior clinoid processes are thick and clubbed, almost closing in the fossa. The air sinuses are invisible: the frontal and nasal bones may be thickened as well as dense. The maxillae may be affected but the mandible almost invariably escapes. Calcification of the dural folds may be seen. The vertebral bodies, if not uniformly dense, usually present two horizontal bands of dense bone above and below, with a less dense band between. The ribs are affected, sometimes irregularly, but the rib cartilages escape. In cases complicated by a tendency to generalised calcinosis, calcification may be seen even in children in the main arteries of the limbs, in the ligaments, the larger tendons, the kidneys, the walls of the trachea, the stomach, and in other soft tissues. Coxa vara, not the result of fracture, may be present: it was seen in three brothers all of whom showed generalised osteopetrosis. The deformity may be of the infantile or cervical type.

Pathological features—The petrosed bone is white-grey on section. The shaft of a long bone may be completely solid with the medullary cavity obliterated. The pathological changes are essentially sclerotic in nature, with increase in number and thickness of trabeculae. The architecture is disorderly and irregular. There may also be hypercalcification of

cartilage with persistence of nodules of calcified cartilage in the zone of newly formed trabeculae. (McCune and Bradley 1934.) The density may be increased further by deposition of calcium salts in the reduced medullary spaces when general calcinosis is also present. Spherical foci of ossification appear in the calcified cartilage. No fatty marrow is to be seen, what marrow there is being myeloid. Osteoblasts are either normal or increased in number: osteoclasts may be normal in number or absent. An important change found in the marrow is fibrosis. It has been suggested—and this seems quite possible—that in some cases the severity of the anaemia depends more on fibrosis of the marrow than on reduction of the marrow space by sclerosis. Reports on the chemistry are confusing, but it seems clear that the ossified bone matrix is normal in composition.

Diagnosis—The diagnosis is usually easy when radiographic examination has once been decided upon. In mild cases, chronic poisoning with lead or phosphorus must be excluded, while in certain localities, even in this country, fluorosis is worthy of consideration. Jupe (1938) reported in two cretins the finding of juxta-epiphyseal dense bands in the metaphyses which disappeared under treatment. Occasionally, limited distribution of the dense bone may give rise to difficulty in classification: such cases are probably best placed in the melorheostosis group.

The writer is greatly indebted to friends who have supplied him with clinical details of cases under their care, and for permission to reproduce some figures from the British Journal of Surgery.

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CASE 14—OSTEOPETROSIS—Severe Type

(Figs. 35–40.) E. L., girl aged 7 years. Genu valgum. Flat feet. Bilateral optic atrophy, left eye practically blind. Toes and fingers clubbed and oedematous. *Blood*—Red cells, 3,912,000. White cells, 5000. Haemoglobin, 47 per cent. Colour index, 53. Right femur fractured by fall and left femur fractured during application of tourniquet. (Under Mr McCrae Aitkin.)

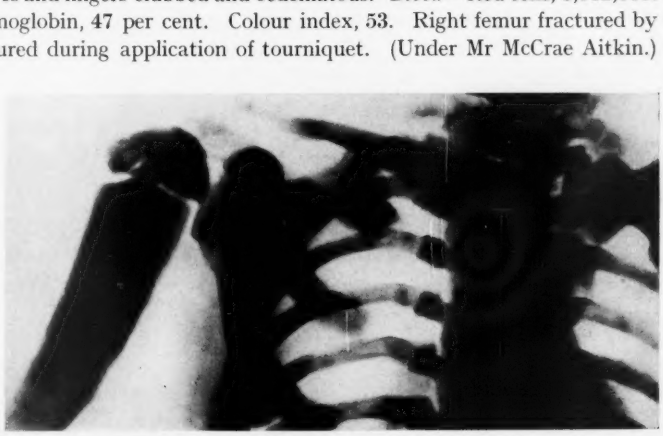


FIG. 35

Case 14. The shoulder, upper ribs, and spine show involvement of all bones in the film. In the humerus there is involvement of the epiphyses; the metaphysis and shaft are enlarged ("clubbed") and dense.

FIG. 36

FIG. 37

FIG. 38

Case 14. The foot (Fig. 36) shows every bone affected, the intensity being most marked in the tarsus, decreasing progressively towards the tips of the toes. Note that the metatarsals show general increased density which is greatest in the metaphyses, whereas in the phalanges the petrosis is almost confined to the metaphyseal regions where the dense bone shows transverse striation. The hand (Fig. 37) shows distribution of petrosis similar to that of the foot. The carpal bones show uniform density. In the region of the knee joint (Fig. 38) there is very abrupt change from the less dense shafts of the femur and tibia to the more dense metaphyses. Note the obvious "clubbing" of these extremities, with a suggestion of cross striation in the tibia and fibula, and some longitudinal striation in the femur. The epiphyses are as dense as the metaphyses.

FIG. 39

FIG. 40

Case 14. The skull (Fig. 39) is somewhat thickened and shows abnormal density especially of the base. There is no sign of the frontal sinuses. The pituitary fossa is rather small and the posterior clinoid processes are clubbed. There is some increased density of the maxillae but not of the mandible. The pelvis and upper femora (Fig. 40) show marked "clubbing" of the femur on each side, and complete lack of structure except the epiphyseal lines. The sacrum is less affected. In the iliac bones, alternate dense and less dense bands are well seen. There is a curious vertical band of less dense bone in the upper part of the necks of both femora.

CASE 15—OSTEOPETROSIS

(Figs. 41–44.) Half-caste Australian girl, aged two and a half years, who had never walked or talked. The parents and one sister were said to be normal. There was a slight squint and advanced optic atrophy. Anaemia and splenomegaly had been present for nearly two years. There was hypotonia of muscles and muscular wasting. The bones were easily palpable and the ends of the long bones were felt to be much enlarged. *Blood examination*—Red cells, 3,000,000. White cells, 10,000. Haemoglobin, 21 per cent. Wassermann negative. Blood calcium and phosphorus normal. (Under Dr Verco of Adelaide.)

FIG. 41

Case 15. The tibiae and fibulae show well-marked clubbing and clear bands, with some longitudinal striation towards both extremities of the bones. The bones of the feet were not well seen but were obviously dense. The upper tibial epiphyses show a clear zone, surrounded by denser bone, and enclosing a dense central area.

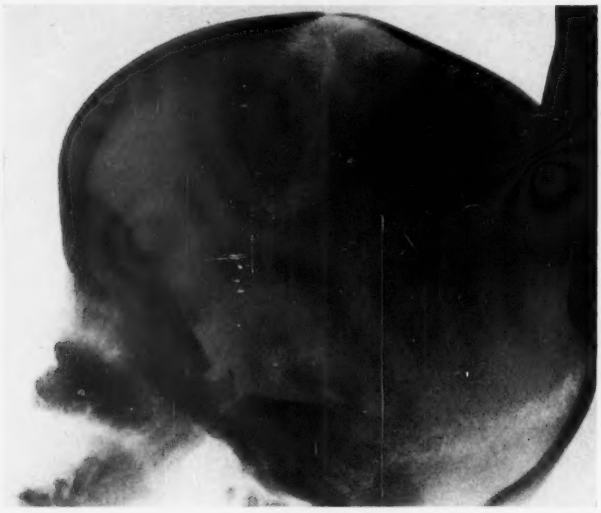


FIG. 42

FIG. 43

FIG. 44

Case 15. The skull (Fig. 42) shows increased density, especially of the base, and a small pituitary fossa with enlarged posterior clinoids. The lower jaw is strikingly normal in density but the maxillae are petrosed. The lower limbs (Fig. 43) show increased density and loss of the normal architecture of all bones. Clubbing of the metaphyses is striking, being most marked at the lower ends of the femora. Clear bands are seen, corresponding roughly with the junction of shafts and metaphyses. The upper tibial epiphysis shows a clear ring encircling and surrounded by denser bone. Clear bands are also seen in the lower thirds of the forearm bones and metacarpals (Fig. 44) accompanied by slight enlargement. There is a healed fracture of the left ulna. The phalanges, as usual, are less affected than the rest of the skeleton, and they display no clubbing.

CASE 16—OSTEOPETROSIS—Benign Type

(Figs. 45–49.) H. R., girl aged six years. Condition inherited from the mother who is mildly affected (Case 17, Fig. 50). There was no history of fractures. The child was admitted to hospital for septic arthritis of the left hip joint. Radiographic investigation showed clear evidence of a benign degree of osteopetrosis. *Blood examination*—Red cells, 4,500,000. White cells, 11,800. Haemoglobin, 75 per cent. Red cells normal. Serum calcium rather high (12·8 mgms.); phosphorus normal (5·6 mgms.). No sign of optic neuritis. (Under the late Sir Henry Gauvain.)

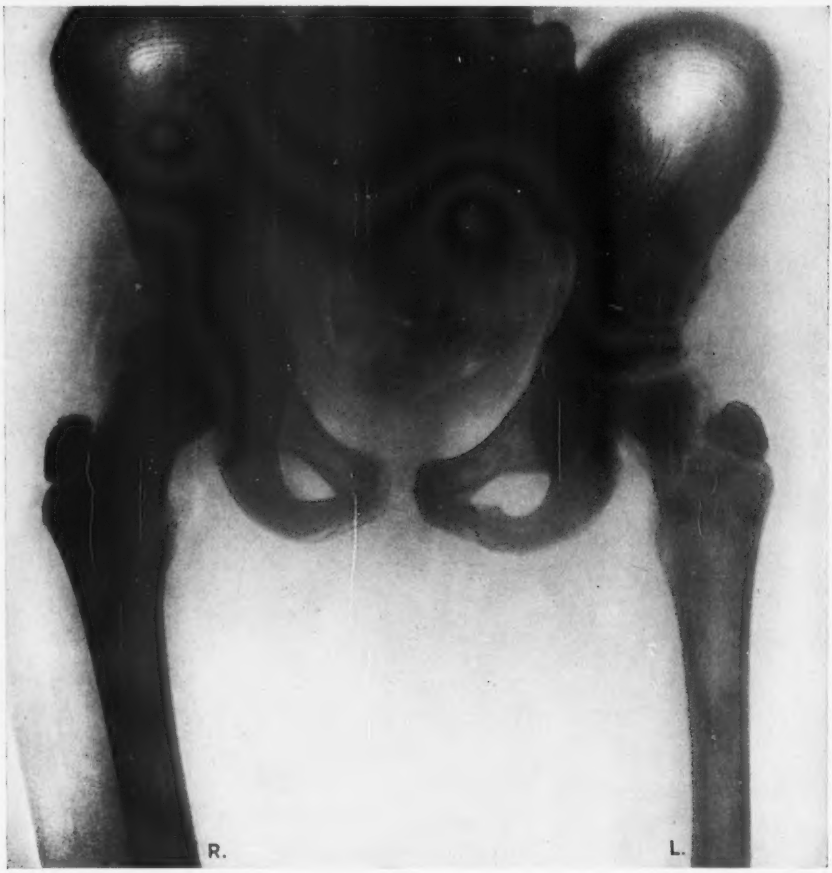


FIG. 45

Case 16. Pelvis and upper femora showing an unusual number of narrow alternating bands parallel to the iliac crests. The femora are both affected in a somewhat patchy and unusual way, the appearance of the upper femoral shafts being rather suggestive of that seen in some cases of diffuse fibrosis of bone. The changes in the region of the left hip joint are due to the infection of the hip joint, with resulting decalcification causing less density of the left side of the pelvis and left femur than on the right side.

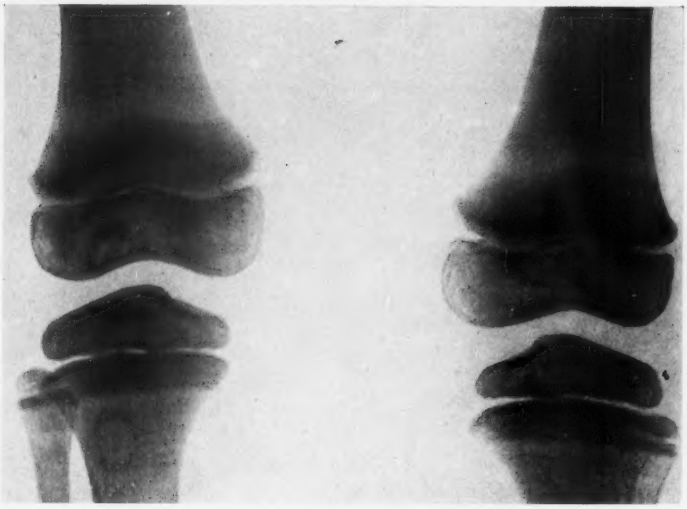


FIG. 46

FIG. 47

FIG. 48

Case 16. The knee joints (Fig. 46) show alternate dark and light transverse bands with greatest density at the ends of the metaphyses suggesting that the tendency to formation of osteopetrotic bone is becoming more marked. The bands are broader and less sharply defined than the so-called "lines of arrested growth." Dense bone in the epiphyses is distributed irregularly. The foot (Fig. 47) shows unusual patchy distribution of dense bone in the metatarsals, typical bands in the phalanges which are parallel with but not adjacent to the epiphyseal lines, and well-marked single or double rings in the tarsal bones. In the hand (Fig. 48) there are dense bands and irregular areas of density in most bones. Note that the cortical bone seems less affected than the endosteal bone. All four inner metacarpals show a dense band at their bases, while only two show density towards the distal or growing ends. Dense bands in the phalanges are some distance from the epiphyseal lines and not in the most recently formed bone as in Fig. 46. The larger carpal bones show single dark circles.

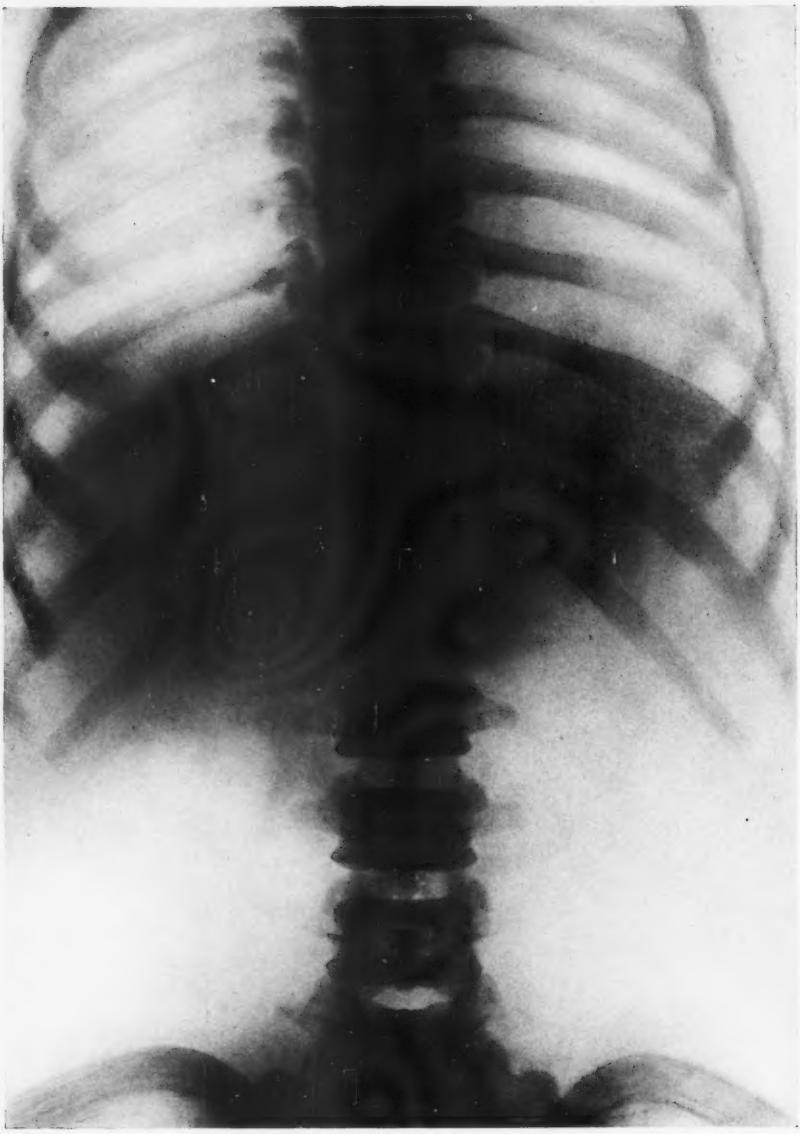


FIG. 49

Case 16. The spine shows clearly the three bands in the vertebral bodies—a lighter band between two dark bands. The ribs show some increased density but they are not strikingly affected.

CASE 17—OSTEOPETROSIS—Mildest Type

(Fig. 50.) Mrs R., mother of patient, Case 16. In perfect health. Dense bone was seen only at or near the ends of long bones. The outer condyle of the one femur that was X-rayed, and the outer tuberosity of the corresponding tibia, were more dense than the inner.

FIG. 50

Case 17. Hand showing similar distribution of dense bone to that seen in her daughter (Fig. 48). The somewhat erratic distribution in the distal parts of the two outermost metacarpals is strikingly similar in the two cases.

CASE 18—OSTEOPETROSIS

(Figs. 51–56.) K. L., woman aged seventy-three years. The bone condition was discovered by accident after admission for a fracture of the femur. Spleen not enlarged. No anaemia. No optic atrophy. Blood examination negative. Right femur had been fractured twice and left femur once. Died at age seventy-five years. (Under Dr W. J. Gill.)

FIG. 51

FIG. 52

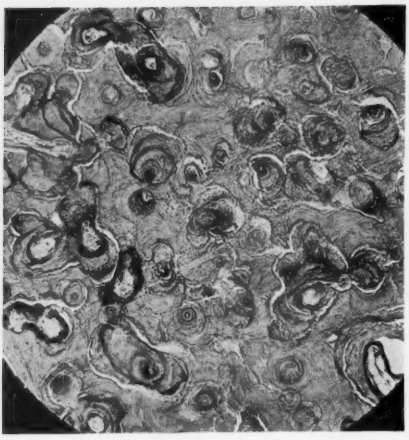


FIG. 53

Case 18. The hands (Fig. 51) show well-marked petrosis of all long bones. Note the alternating bands in the proximal phalanges. The lower epiphyses of radius and ulna and the carpal bones are partly involved. Cross section of the shaft of the femur (Fig. 52) shows complete obliteration of the medullary cavity (the dark patch is an artefact). Microscopic section of the femur (Fig. 53) shows the curious architecture of the dense bone. (By courtesy of Professor W. G. Barnard.)

FIG. 54

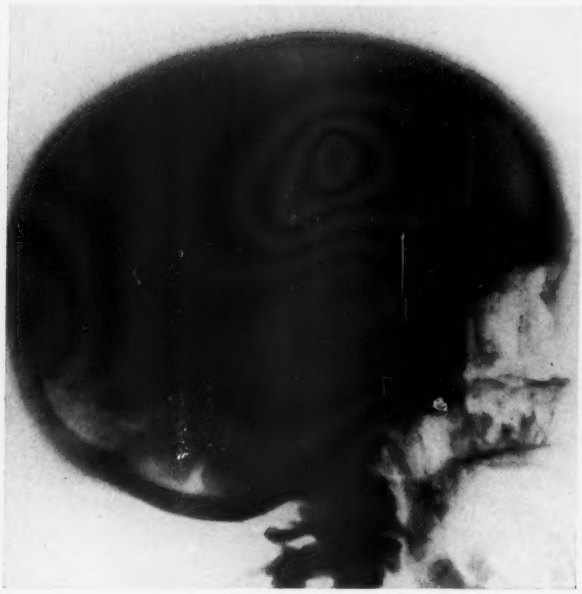


FIG. 55

FIG. 56

Case 18. The pelvis (Fig. 54) shows generally increased density to a marked degree; there are no definite curved bands in the ilia. Old fracture neck of right femur. The skull (Fig. 55) shows increased density particularly at the base. The pituitary fossa is slightly small. The frontal sinuses are not visible. The density of the facial bones and lower jaw is apparently normal. The cervical vertebrae are dense. The lower limb (Fig. 56) shows almost complete obliteration of structure of all bones including the patella. There is arthritic lipping of the knee joint.

CASE 19—OSTEOPETROSIS

(Figs. 57–61.) C. R., boy aged nineteen months. Admitted to hospital because of general backwardness and failure to gain weight. Right facial palsy. No history of fractures. *Blood examination*—serum calcium 13·5 mg. per cent. Blood cholesterol, 162 mg. Haemoglobin, 84 per cent. Wassermann and Kahn reactions negative. (Under Professor Alan Moncrieff.)

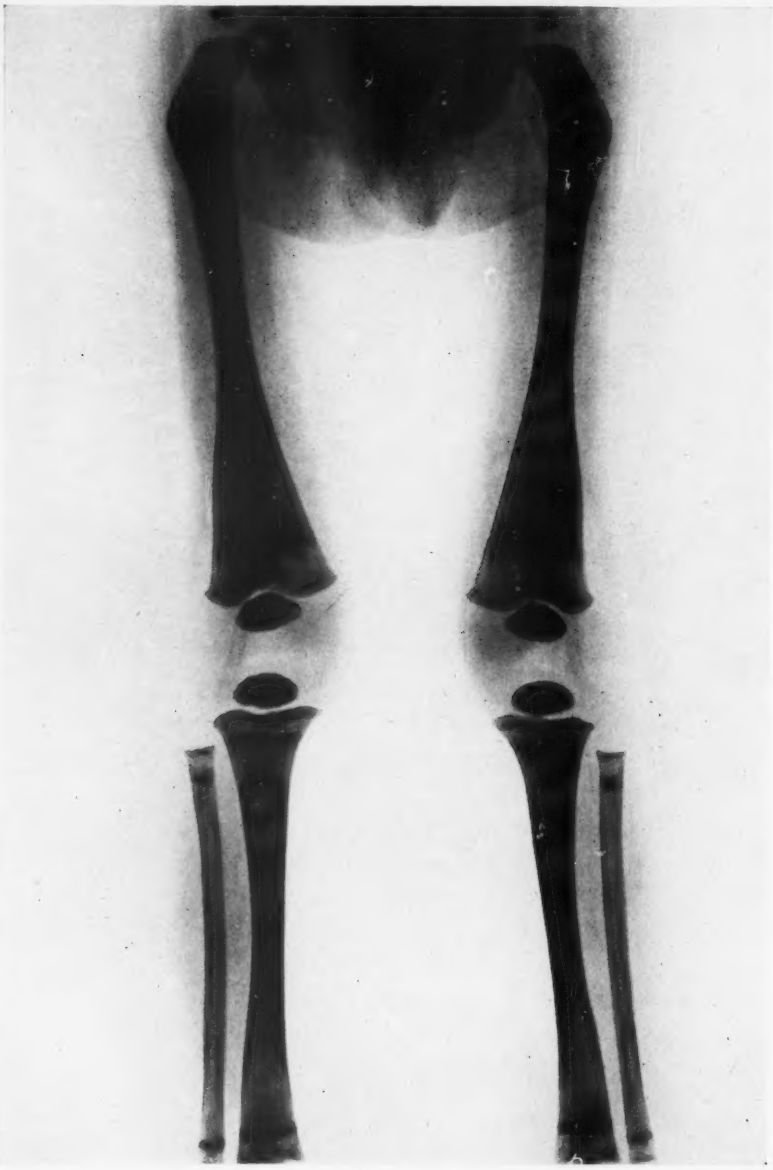


FIG. 57

Case 19. November 1945. The lower limbs show general increased density of long bones ending abruptly above and below in intensely opaque bands a short distance from the epiphyseal lines. Recently formed bone in the metaphyses is distinctly less dense than in the rest of the shafts.

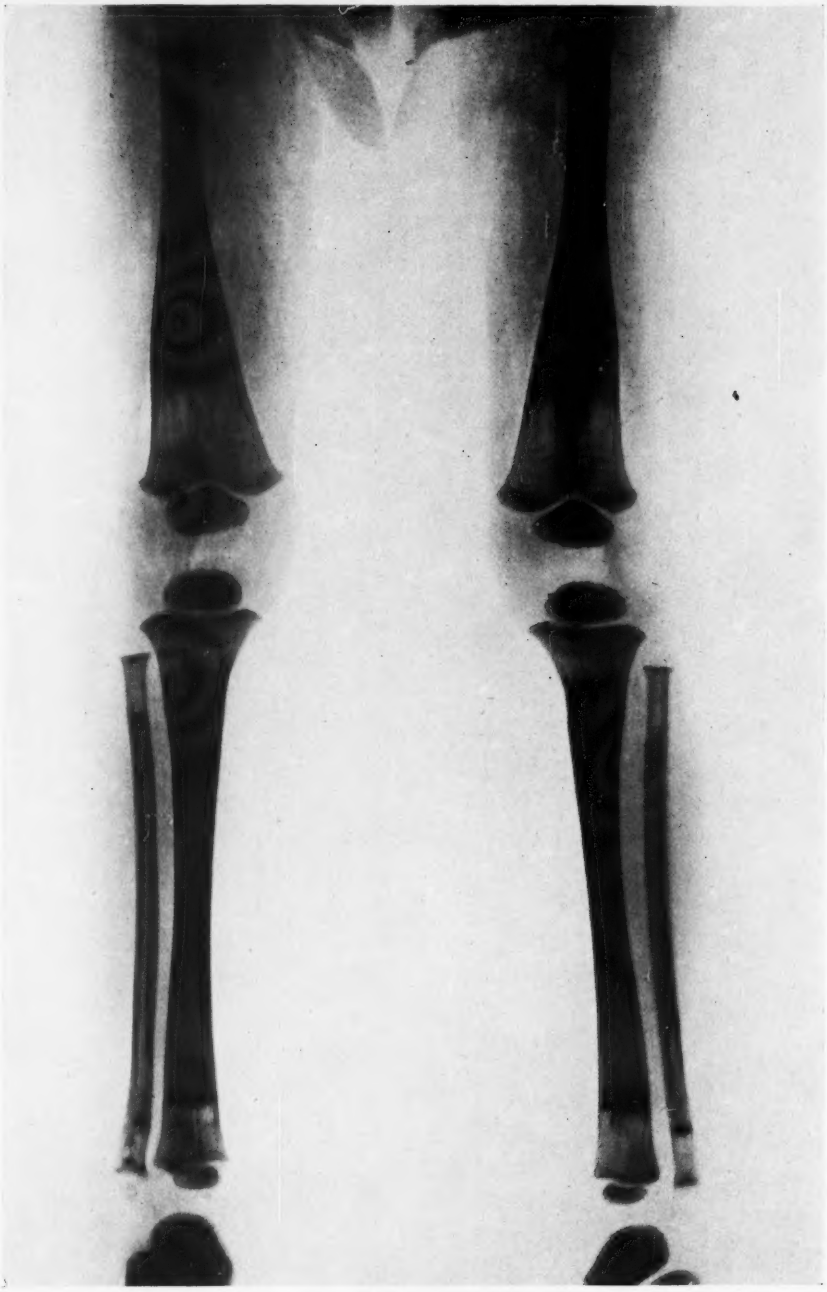


FIG. 58

Case 19. July 1946. Film taken for comparison with Fig. 57. Note that bone added during the interval of eight months has been unaffected by petrosis—in fact it is somewhat porotic. New bone formed round the abnormally dense epiphyseal nuclei is of normal density. The same is true of the tarsal bones.

FIG. 59

FIG. 60

FIG. 61

Case 19. July 1946. Radiographs of the upper limbs (Fig. 60) show similar distribution and extent of density as in the lower limbs. The skull (Fig. 59) shows increased density, notably of the base, with thickening in the frontal region. The pituitary fossa is small. The maxillae are affected. There is increased density of vertebrae, sacrum, ribs and sternum (Fig. 61).

CASE 20—OSTEOPETROSIS

(Figs. 62–65.) R. P., boy aged five years. Admitted to hospital for pain in the region of the left hip joint of some months' duration. No optic neuritis. *Blood examination*—Red cells, 4,360,000. Haemoglobin, 90 per cent. White cells, 2,600. (Nine days later 6000.) Neutrophils, 40 per cent. Lymphocytes, 56 per cent. Chemical analysis showed no abnormality. The evidence suggested an atypical case of osteopetrosis. (Under Mr D. B. Craig.)

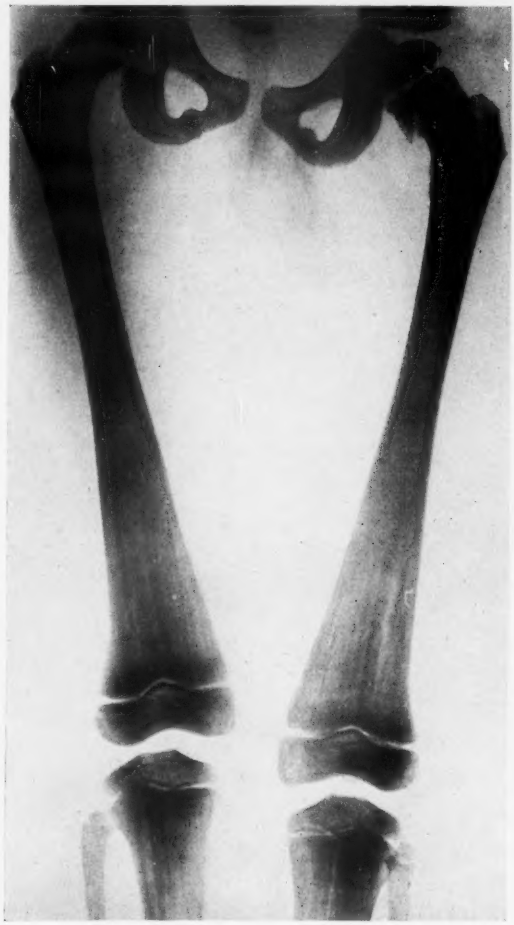


FIG. 62

FIG. 63

Case 20. The shafts of the femora (Fig. 62) show unusual distribution of increased density particularly in the region of the knee joints. There is marked longitudinal striation of the metaphyses. Note what appears to be a chip fracture of the neck of the left femur with minimal displacement of the head, suggestive of the infantile type of coxa vara. The spine (Fig. 63) shows abnormal density of the upper and lower parts of each body with a less dense band between, which is strong evidence in favour of the diagnosis of osteopetrosis.

FIG. 64

FIG. 65

Case 20. The skull (Fig. 64) shows abnormal density particularly of the base. The pituitary fossa is small. The upper, and possibly the lower, jaw seem to be affected. The pelvis and hips (Fig. 65) show fairly generalised osteopetrosis. There are faint bands parallel with the crests. The fracture of the neck of the femur, suggestive of infantile coxa vara, is again shown in this film.

John Hunter

John Hunter was born on February 13, 1728, at Long Calderwood, a small estate in Lanarkshire about seven miles from Glasgow. His father, whose name was also John, was one of the Hunters of Hunterston in Ayrshire—an old Scottish family. He and his wife Agnes had ten children of whom William was the seventh, and John the youngest. Both brothers achieved fame as anatomists but they were very dissimilar in temperament and character.

William was diligent as a boy, and at the age of fourteen went with a bursary to Glasgow University where he studied for five years. Afterwards he spent a short time in Edinburgh, and finished his medical training at St George's Hospital, London. Later he started a school of anatomy in Covent Garden which soon acquired considerable reputation by reason of the facilities he offered for dissection, and his own capacity as a lecturer. In 1770 he transferred the school to Great Windmill Street where a building had been erected with lecture theatre, dissecting rooms, and museum. He had already been elected physician-accoucheur to the Middlesex Hospital and later was appointed Physician Extraordinary to Queen Charlotte; but his main interest was anatomy, and he lectured upon it to the end of his life. He was elected a Fellow of the Royal Society to whose transactions he contributed a paper "On the Structure and Diseases of Articulating Cartilages." In this communication he was the first to give a clear account of the structure and arrangement of synovial membranes.

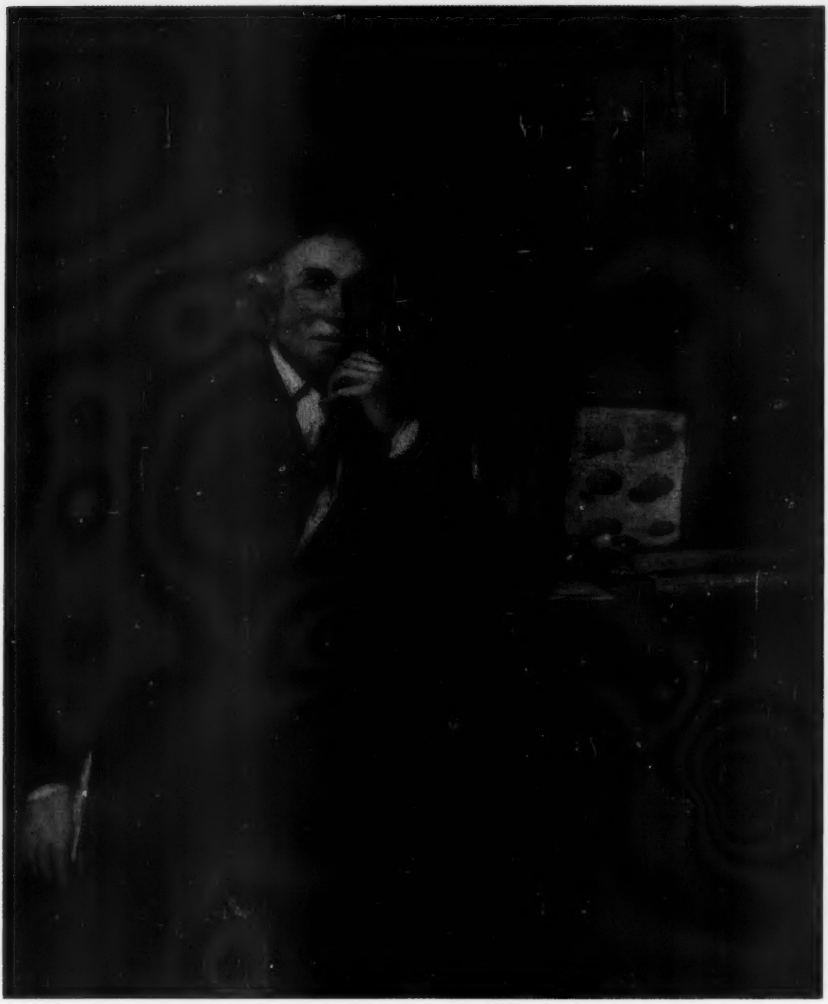
John Hunter was ten years younger than William Hunter. In early life he had none of the studious habits of his elder brothers, both of whom went to a university, one studying medicine and the other law. Being the youngest, and favoured by his mother, John was somewhat undisciplined. He was averse to schooling of any kind but gave early evidence of the thread of his peculiar genius when he rambled in the woods, watched ants, bees, birds, and tadpoles, and pestered country folk with simple questions on natural history. Until the age of twenty his mind remained fallow and untroubled, but on the verge of manhood he woke from slumber and for the next forty-five years worked so prodigiously in the production and study of scientific material in medicine and biology that the like of him has not been seen again. He began by joining his brother in the school of anatomy where he arrived exactly 200 years ago. He soon acquired such patience and skill as a dissector, and such knowledge of anatomy, that within twelve months he was appointed demonstrator. He studied surgery under Cheselden, and afterwards under Pott, the two master surgeons of the day. Later he enrolled as a pupil at St George's Hospital, and in due time became house surgeon. In 1759, through overwork in the dissecting rooms, he developed pneumonia; symptoms appeared which were suggestive of tuberculosis. He therefore sought a change of work and secured appointment as a staff-surgeon in the army. He sailed with the expedition to Belleisle, and in the war with Spain served on the frontier of Portugal. This gave him extensive experience of gun-shot wounds which was embodied in his "Treatise on the Blood, Inflammation, and Gun-shot Wounds."

He left the army after two years' service, took a house in Golden Square, and began his career as a surgeon. Having spent twelve years in the dissecting room, and carried out many researches, he brought to the practice of surgery a mind trained in scientific investigation. Private practice was subordinated to the study of comparative anatomy and biology which demanded all the time and money that he could devote. He acquired specimens from the menageries—dissecting the bodies of animals that died; and in order to study living animals both by experiment and by observation of their habits he bought, in 1764, two acres of land at Earl's Court which at that time was in the country, two miles beyond London. There he organised an experimental station and housed a great variety of wild and domestic animals

from all over the world. He studied the structure of innumerable living organisms and observed the effects of disease and injury upon it. Dissected specimens were preserved carefully, and thus was built up an anatomical and pathological museum which became the bedrock of scientific study of surgery in England. He was elected a Fellow of the Royal Society in 1767 and was awarded its Copley Medal. In the next year he was elected surgeon to St George's Hospital and soon afterwards became a member of the Corporation of Surgeons. The pupils he attracted, who afterwards achieved great distinction, included Edward Jenner, Abernethy, Cline, Earle, and Astley Cooper. Unlike other teachers his lectures on surgery were based upon rational pathology; he insisted that the principles of surgery must first be understood before the cause of disease could be appreciated. But he had none of the eloquence of his brother; extempore expression of the thoughts which surged within him did not come easily. He read almost entirely from manuscript and even then his language was laboured. But it was what John Hunter had to say that was important rather than his manner of saying it. The casket was unadorned but the treasure within was of great value.

Soon after election to the staff of St George's Hospital he moved to a house in Jermyn Street, previously occupied by his brother, which was nearer to hospital, better situated for private consulting work, and offered greater accommodation for his increasing collection. A few years later he married Anne Home, a lady noted for her beauty, wit, and accomplishment—a social figure in the world of art. She wrote the words for Haydn's " Creation " and the stanzas for his English canzonets, the best known of which begins with the words " My mother bids me bind my hair." They lived in Jermyn Street for fourteen years until the ever-growing museum overran the house and forced Hunter to seek still more spacious accommodation. In 1785 he moved to his last abode, a large house on the east side of Leicester Square. He bought another house close by and, on the land between, built a suite of rooms and a great museum. In his new quarters the dissection and collection of natural history specimens went on apace. At the same time his practice grew to such an extent that on the death of Percivall Pott in 1788 Hunter succeeded him as the first surgeon in England. In his attention to patients he was thoughtful and self-sacrificing; he gave his services free to non-beneficed clergy and struggling artists.

Hunter took a conservative view of operations and always aimed at diminishing their severity, regarding many of them as an imperfection of the art of healing. He was deeply impressed by the natural resistance of the body to disease, and by its struggle against anything impairing the function of one of its parts. The sole business of the surgeon was that of a helper, claiming no more for his operation than lending his aid in that resistance and that struggle. It was appreciation of this inherent power of living tissues which led to his classical operation for popliteal aneurism. He was alarmed at the high mortality of prevailing treatment by amputation, or double ligature with evacuation of the blood sac. He had tied one of the external carotid arteries of a stag in order to observe the effect upon growth of the corresponding antler; to his surprise the horny outgrowth still kept growing. Wondering what had happened to his ligature he had the animal killed for inspection and to his amazement Hunter found that small branches of the artery above and below the occlusion had enlarged under " the stimulus of necessity," and by their anastomoses had restored blood supply to the growing part. He argued that if in the deer, collateral vessels could maintain circulation after ligation of the main artery, the same should happen in the human lower limb after occlusion of the femoral artery by single ligature. Acting upon this deduction, in 1785, he tied the femoral artery in the lower part of the thigh in a patient at St George's Hospital with a popliteal aneurism. Within six weeks the man left hospital with his life and his limb. The fibro-muscular sheath through which the femoral artery passes has ever since been known as Hunter's Canal. The evolution of this operation illustrates John Hunter's scientific approach and appeal to experimental methods in seeking the solution of surgical problems—an approach which in his day was strange, no matter how familiar it may be in ours.



John Hunter

1728-1793

"For their work continueth. And their work continueth,
Broad and deep continueth. Greater than their knowing."

RUDYARD KIPLING.

An investigation of great interest to orthopaedic surgeons was prompted by a personal injury. In 1767, while dancing, he ruptured his tendo Achillis. Thereupon he studied in animals the manner of reunion of divided tendons. " He divided the same tendon in several dogs, by introducing a couching needle under the skin at some distance from it, and killed the dogs at different periods to see the progress of the union, which was found to be similar to that of fractured bones where the skin is not wounded." His museum included fine specimens of tenotomy from the ass and deer. The ends of the divided tendons had retracted but " the uniting medium was not distinguishable from the tendon itself except by being less glistening, by its fibres being less regularly parallel and longitudinal, and by its surfaces being united with the surrounding fibrous textures." Hunter observed that repair of the tendons proceeded just like that of simple fractures, and without suppurative inflammation. This was an important statement of a principle. Upon it was founded the practice of subcutaneous surgery which until the coming of Lister was the only safe operative procedure for the cure of deformities.

He attempted the transplantation of tissues and successfully implanted a sound human tooth into a cock's comb. This striking experiment demonstrated his zeal and patience, for it was accomplished only after many failures. In the remarkable specimen preserved in his museum could be seen the injected blood-vessels of the comb penetrating the tooth. He thus proved that tissues of low metabolism could be grafted elsewhere if the blood supply was adequate—a principle upon which depended the successful bone grafting of later days. Bone growth aroused his interest. After much experimental work on pigs he laid it down that long bones are lengthened from the ends near the epiphyses, and not by interposition of new bone in the interstices of the old. Furthermore he showed that a bone became thicker by the deposition of new matter subperiosteally; and that bone is constantly undergoing change with simultaneous absorption and deposition of osseous tissue—an observation confirmed later by the microscope.

Study of the structure and function of the human body was only part of Hunter's work. His labours covered the whole field of natural history. In what does life consist was a question often in his mind. He regarded it as a principle tenaciously held and independent of structure. He sought to unfold the various phases of life. In his quest he dissected over 500 different species of animals and numerous varieties of plants. His search ranged from the spermaceti whale, with its aorta a foot in diameter into which passed at each heart-beat twelve gallons of blood, to the life of the bee which he watched for twenty years. His study of the structure and of the social habits of this honey-making insect was so extensive and protracted that it was only close to the end of his life that he collected his records together for publication in the Transactions of the Royal Society. If this had been his only contribution to knowledge his name would have been memorable.

Although later years were handicapped by ill-health he never slackened in the pace of his scientific work. His day started at six o'clock in the morning and ended well after midnight. For the last twenty years of his life he drank no wine. In 1785 he had his first attack of angina, and for eight years suffered periodical prostration, his life at times being held precariously in the balance. During this period he was well aware that emotional excitement might bring on a cardiac catastrophe. On October 16, 1793, he attended a Board Meeting at St George's Hospital. In a discussion about the admission of pupils to the hospital his feelings were roused. His old malady was stirred to activity. He rose from the table, hoping to control his symptoms, but had hardly reached another room when he fell groaning into the arms of a colleague and died. He was buried in the vaults of St Martin's-in-the-Fields. In 1859 Frank Buckland spent sixteen laborious days searching for John Hunter's remains. At last he found them. On March 28 the same year they were buried with great honour in Westminster Abbey.

John Hunter dedicated his life to scientific investigation and the building of a great museum. He gathered together thousands of his own dissections; even in his last years his

assistant tells how he often saw him " standing like a statue for hours over some delicate bit of dissection." Well has it been said that " he made his name immortal by the labour of his own hands outside the sphere of surgery." The care of the collection, which contained over 13,000 specimens, caused some anxiety to Hunter's family but in young Clift his assistant they found a faithful and devoted guardian. For seven years he kept watch, preventing deterioration in the soft specimens, and making copious extracts from the manuscripts. On June 13, 1799, the Government bought the collection for £15,000 and transferred it to the care of the Corporation of Surgeons. A charter was granted to the Corporation on March 22, 1800, whereby they were constituted the Royal College of Surgeons in London and were empowered to examine candidates for the Membership. The surgeons gave up Surgeons' Hall and moved to a house in Lincoln's Inn Fields. In 1806 Parliament granted £15,000 to the College to build a museum and three years later another grant of £12,500. The surgeons themselves spent over £21,000 of their own money. By a charter of 1843 the title of the College was changed to the Royal College of Surgeons of England and the Fellowship of the College was instituted.

John Hunter has exercised a profound influence on British surgery. This was achieved not by social gifts or personal attraction but entirely by his scientific mind. When he started dissecting, three years only had passed since surgeons had ceased to be associated formally with the " art and mystery of barbers." But through his immense labours in comparative anatomy, physiology, and pathology, he raised the status of surgery to that of a scientific profession. In his quest for truth by observation and experiment he displayed a penetrating vision extending far beyond the horizon of his own time. His country experimental station long anticipated " Down House " which is now the experimental farm of the Royal College of Surgeons. Most of his teaching is inevitably bound in the corpus of surgical doctrine and has lost its identity. Like Lister he was interested in the phenomena of inflammation and coagulation of the blood but he was denied the use of a microscope and he little dreamt of a world of micro-organisms. But his keen intellect noted and stressed mysterious variation in the reaction of tissues to injury according to whether the skin was broken or unbroken. If the skin was intact the healing process proceeded smoothly; whereas with a broken skin suppuration was the rule and repair was disturbed and delayed. And he pondered why. His great museum is the proud heritage of the Royal College of Surgeons of England and it is fitting that on the anniversary of his birthday an oration in praise of him should be delivered by a distinguished disciple.

ARTHUR ROCYN JONES

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PIONEERS OF OSTEOGENY

JOHN HUNTER : 1728-1793

JESSIE DOBSON, LONDON, ENGLAND

From the Department of Human and Comparative Anatomy, Royal College of Surgeons of England

Hunter made observations on the growth of bone when he first began his lectures in 1772 but his experiments to show the method of growth were probably made long before this date. He was endeavouring to prove two hypotheses: 1) that growth takes place by the addition of new bone on the surface; and 2) that the rate of growth is not uniform in all parts of the bone. Two different methods were used. Original specimens of each type of experiment are still preserved in the Museum of the Royal College of Surgeons of England and are here illustrated. In both methods Hunter had been anticipated by Henri Louis Duhamel du Monceau (1700–82).

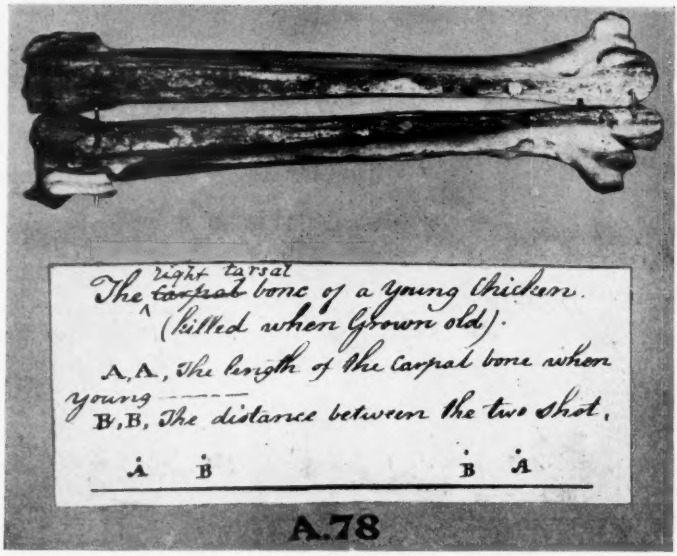
The right tarsal bone of a young Chicken.
(killed when Grown old).
A,A, The length of the Carpal bone when young
B,B, The distance between the two Shot.
A B B A
A.78

FIG. 1

Photograph of one of John Hunter's specimens, with the original note, illustrating his experiments in the investigation of bone growth by the insertion of lead shot into drill-holes.

Though Hunter left no consecutive description of his many experiments on bone growth, extracts from his writings give a clear indication of his methods and results: " Bones do not grow by having new particles put into the interstices of previously formed parts, so as to remove these to a greater distance from each other, by which means they should grow larger —as, for instance, if I put a sponge into water, the water getting into all the interstices makes

it larger—but they grow by the addition of new bone on the external surface. I took a pig of a very large breed when young, bored two holes in the tibia, and put a shot into each, measuring on a card the distance of each from the other. I allowed this pig to grow up to its full size, then killed it and took out the bone, and I found the two holes exactly the same distance from one another as at first. Now if the bone had grown in all its parts, these two shot would have been removed to a distance from each other proportionate to the growth of the bone."

A similar experiment was made on the right tarso-metatarsus of the domestic fowl and Fig. 1 is a photograph of the actual specimen. It also shows the original note of the experiment which is preserved in the jar. Fig. 2 is a photograph of another of Hunter's specimens in which he used a different method to demonstrate the same thesis—that bone does not grow at the same rate throughout. This preparation is the left tarso-metatarsus of the domestic

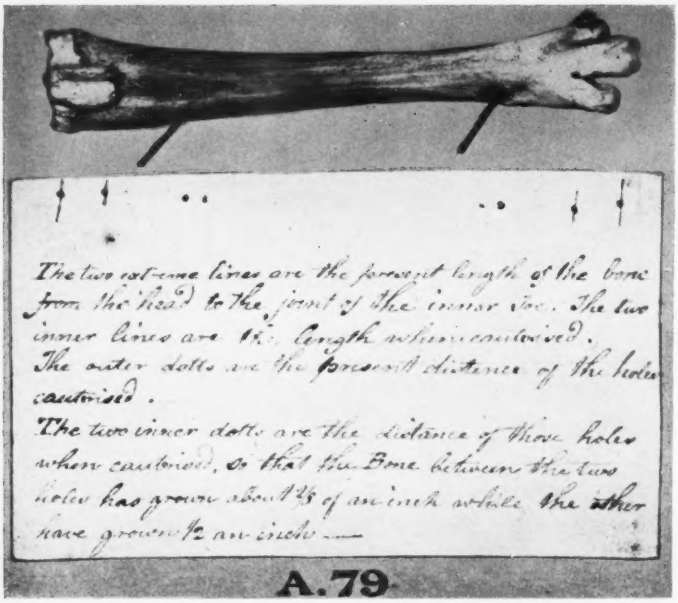
The two outermost lines are the present length of the bone from the head to the joint of the inner toe. The two inner lines are the length when cauterised.
The outer dotts are the present distance of the holes cauterised.
The two inner dotts are the distance of those holes when cauterised. So that the Bone between the two holes has grown about 1/8 of an inch while the other have grown 1/2 an inch —
A.79

FIG. 2

Photograph of another Hunter specimen, preserved in the Royal College of Surgeons of England, in which he determined the method of bone growth by measuring the distance between points of cauterisation.

fowl and here he was able to prove that increase of bone distal to the points of cauterisation is more than double that of the space included between them. Here again the original note on the experiment is preserved in the jar.

The second group of experiments consisted in feeding animals with madder, which has the property of staining more vividly with a red colour that part of the bone which is added while the animal is being fed with food mixed with madder. Hunter's best description of these experiments is to be found in that section of his work which deals with growth of the teeth: " Take, for example, any young animal, as a pig, and feed it with madder for three or four weeks; then kill the animal, and upon examination you will find the following

appearances: first if this animal had some parts of its teeth formed before the feeding with madder, those parts will be known by their remaining of the natural colour; but such parts of the teeth as were formed while the animal was taking the madder will be found to be of a red colour. This shows that it is only those parts that were formed while the animal was taking the madder that are dyed; for what were already formed will not be found in the least tinged. This is different in all other bones, for we know that any part of a bone which is already formed is capable of being dyed with madder, though not so fast as the part that is forming: therefore, as we know that all other bones when formed are vascular, and are thence susceptible of the dye, we may readily suppose that the teeth are not vascular, because they are not susceptible of it after once being formed. But we shall carry this still further: if you feed a pig with madder for some time, and then leave off for a considerable time before you kill the animal, you will find the above appearances still subsisting, with this addition, that all the parts of the teeth which were formed after leaving off feeding with the madder will be white. Here then in some teeth we shall have white, then red, and then white again; and so we shall have the red and the white colour alternately through the whole tooth. This experiment shows that the tooth, once tinged, does not lose its colour: now as all other bones that have been tinged lose their colour in time, when the animal leaves off feeding with madder (though very slowly), and as that dye must be taken into the constitution by the absorbents, it would seem that the teeth are without absorbents as well as other vessels. This shows that the growth of the teeth is very different from that of other bones. Bones begin at a point, and shoot out at their surface, and the part that seems already formed is not in reality so, for it is forming every day by having new matter thrown into it, till the whole substance is complete and even then it is constantly changing its matter." Fig. 3 illustrates these madder experiments and is a photograph of the lithograph made from the original drawing prepared for Hunter by his assistant, William Bell. The original drawing is preserved in the library of the Royal College of Surgeons.

FIG. 3

Photograph of William Bell's lithograph illustrating John Hunter's madder experiments.

Mention of "absorbents" in the above extract brings us to Hunter's original conclusions concerning bone and bone growth: "Since we know that bones do not grow by fresh matter being put into all parts, so as to push the old matter to a greater distance but by new matter laid upon the external surface, let us see whether it is possible for bone to grow and retain its form without being taken down. Let Fig. 3, Plate XX, represent the head of the thigh bone of a foetus. Now if the increase was accomplished by superimposed layers, one over the other, as in Fig. 4, the head of the bone would necessarily become of an enormous size and in time would come down so as to occupy the middle of the bone while the cavity in the centre would not be enlarged. Absorption, therefore, must necessarily go on to keep the bone of its proper shape. Hence I call this the modelling process, for without this combined

action of absorption and deposition the animal could not grow as it now does." The original figures to which Hunter alludes are reproduced in Fig. 4.

In addition to his experiments conducted to show the actual way in which increments were added to growing bone, Hunter (in Owen's "Essays and Observations") made this

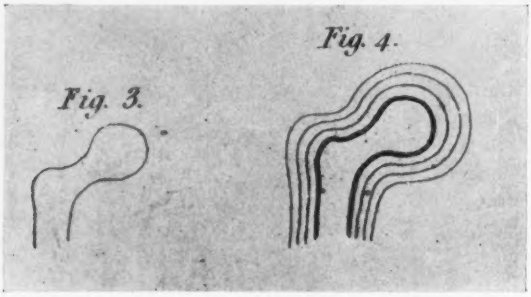

FIG. 4

Plate XX of John Hunter's works illustrating the principle of the "modelling process."

shrewd, and doubtless original, observation on the tissue basis from which bone could be developed: "Bone is not the original skeleton in any animal, but only of the adult; for in the first formation of any animal, which afterwards is to have bone, the skeleton is either membrane or cartilage, which is changed *for* bone, but not *into* bone."

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ORTHOPAEDIC SURGERY IN THE SIXTEENTH AND SEVENTEENTH CENTURIES

1. Luxations of the Shoulder

WILLIAM BROCKBANK and D. LL. GRIFFITHS, MANCHESTER, ENGLAND

From the Medical Library of the University of Manchester

Many books on surgery of the sixteenth and seventeenth centuries include important and well-illustrated sections on dislocations, which is in rather striking contrast with the much smaller space allotted to fractures. These accounts, and the woodcuts that accompany them, are copied with slight modifications from book to book and from century to century. They give a clear account of the methods employed at that time. The illustrations reproduced are taken from books published in 1544, 1607, 1625, 1678, and 1693.

Perhaps the standard mediaeval treatise on the surgery of injury was that of Ambroise Paré (1510–1590) whose collected works were published in Paris in 1575. An English translation published in 1678 gives a good example of the text of such works and can speak for itself.

" OF A DISLOCATED SHOULDER "

" The shoulder is easily dislocated, because the ligaments of its dearticulation are soft and loose, as also for that the cavity of the shoulder-blade is not very deep; and besides it is every where smooth and polite, no otherwise than that of the shoulder-bone, for that it is herein received. Add hereunto that there is no internal ligament from bone to bone, which may strengthen that dearticulation, as is in the leg and knee. Wherein notwithstanding, we must not think Nature defective but rather admire God's providence in this thing; for that this articulation serves not only for extension and bending, as that of the elbow, but besides, for a round or circular motion, as that which carries the arm round about, now up then down, according to each difference of site. The shoulder-bone may be dislocated four manner of ways, upwards, downwards, or into the arm-pit, forwards and outwards, but never backwards. Now Hippocrates saith that he hath only seen one kind of dislocation of this bone, to wit, that which is downwards to the arm-pit; and certainly it is the most usual and frequent, wherefore we intend to handle it in the first place. When the shoulder is dislocated downwards into the arm-pit a depressed cavity may be perceived in the upper part of the joint; the acromium of the blade shows more sharp and standing forth than ordinary, for that the head of the shoulder bone is slipt down and hid under the arm-pit, causing a swelling forth in that place; the elbow also casts itself (as it were) outwards and stands further off from the ribs, and though you force it, yet can you not make it to touch them; the patient cannot lift up his hand to his ear on that side, neither to his mouth nor shoulder. Also this arm is longer than the other. Lastly, the patient can move his arm by no kind of motion without sense of pain by reason of the extended and pressed muscles, some also of their fibres being broken."

" Of the first manner of setting a shoulder which is with one's fist "

" First let one of sufficient strength, placed on the opposite side, firmly hold the patient upon the joint of the shoulder: then let another taking hold of his arm above the elbow so draw and extend it downward that the head thereof may be set just against its cavity hollowed in the blade bone. Then let the surgeon lift and force up with his fist the head of

FIG. 1

Reduction of a dislocated shoulder by means of a towel around the operator's neck. It is presumed that reduction over the fist has failed. The resemblance of this method to methods still advocated for "difficult" hip-dislocations is striking. (From the English translation of Paré, 1678.)

FIG. 2

Reduction around the operator's heel (the classical "Hippocratic" method). From de Cruce, 1607. Note again the use of the towel to provide lateral traction.

the bone into its cavity. But if the luxation be inveterate and the hand cannot serve then must the patient's shoulder be fastened to a post with a ligature or else committed to one's charge who may stand at his back and hold him fast. Then the arm shall presently be tied about a little above the elbow with a fillet, whereto a cord shall be fastened; which being put or fastened to a pulley shall be drawn or stretched forth as much as need shall require. Lastly the surgeon with a towel or such-like ligature fastened about his neck and hanging down and so put under the patient's arm-pit near to the luxation, shall by raising himself upon his feet with the whole strength of his neck lift up the shoulder, also at the same time bringing his arm to the patient's breast shall set the head of the shoulder bone." (Fig. 1.)

" Of the second manner of restoring a shoulder ; that is with the heel when the patient by reason of pain can neither sit nor stand "

" The patient must be laid with his back on the ground upon a coverlid or mat and a clew of yarn or leathern ball stuffed with tow or cotton of such bigness as may serve to fill up the cavity, must be put under his arm-pit, that so the bone may straightways the more easily be forced by the heel into its cavity. Then let the surgeon sit beside him even over against the luxated shoulder, and if his right shoulder be luxated, he shall put his right heel to the ball which filled up the arm-pit, but if the left, then the left heel: Then let him forthwith draw toward him the patient's arm, taking hold thereof with both his hands, and at the same instant of time strongly press the arm-pit with the heel. While this is in doing one shall stand at the patient's back, who shall lift up his shoulder with a towel and also with his heel press down the top of the shoulder blade, another also shall sit on the other side of the patient who, holding him, shall hinder him from stirring, this way or that way, at the necessary extention in setting it." (Fig. 2.)

" The third method of restoring a shoulder "

" Someone who is of competent height and strength shall put the sharp part of the top of his shoulder under the patient's arm-pit and also at the same time shall somewhat violently draw his arm towards his own breast so that the patient's whole body may as it were, hang thereby. In the meantime another, for the greater impression, shall lay his weight on the luxated shoulder shaking it with his whole body. Thus the shoulder, drawn downwards by the one which stands under the armhole, and moved and shaken by the other who hangs upon it, may be restored into its seat." (Fig. 3.)

" Of the fourth method of restoring a dislocated shoulder "

" You must take a perch or piece of wood (somewhat resembling that which the water-bearers of Paris use to put on their shoulders), some two inches broad and some six foot long. In the midst thereof let there be fastened a clew of yarn or ball of sufficient bigness to fill up the cavity of the armhole. Let there be two pins put in it, one on each side of the ball, each at like distance therefrom with which as with stays, the shoulder may be kept in, and upon the ball that it slip not away from it. Let two strong men taller than the patient by nature or art put this perch upon their shoulders. Then let the patient put his arm-pit upon that place where the ball stands up. The surgeon must be ready to pull his hanging arm downwards. Thus the patient as it were hangs on the perch with his shoulder and so the head of the bone shall be forced into the cavity." (Fig. 4.)

FIG. 3

Reduction over the operator's shoulder. The use of a child as an additional traction-force is not classical. The illustration (from the Venice edition of Galen, 1625) does not show the shoulder being shaken with the assistant's "whole body" as in Paré's text.

FIG. 4

Reduction over a wooden yoke. The author's insistence on adequate padding of the wood perhaps helped to reduce the chances of damage to the neuro-vascular bundle. (From Paré, 1678, *loc. cit.*)

FIG. 5

Reduction over the rung of a ladder. This illustration shows the method approved by Paré, one rung of the ladder having been removed to ensure that the patient's head shall not be trapped in his fall. From de Cruce, 1607.

"Of the fifth manner of putting the shoulder into joint which is performed by a ladder"

"You may also restore a shoulder dislocated into the arm-pit, by the help of a ladder after this manner. Let some round body as a ball of yarn which may serve to fill the arm-pit be fastened upon one of the upper steps of a ladder; at the foot of the ladder set a low

FIG. 6

Reduction over the rung of a ladder. This illustration, which appears in Vidius, 1544, shows how *not* to do it.

stool, whereupon let the patient mount, then bind both his legs and also his sound arm behind his back, lest when you are about your operation he hinder and spoil all you do by laying his hand or setting his foot upon the ladder. Then let his arm be presently put over the step of the ladder and his arm-pit put upon the there fastened ball; the patient in the meanwhile being wished to come with his whole body as near into the steps as he is able; for otherwise besides that there is no other method of restoring the luxation there would be no small danger of breaking the shoulder bone. Also let him take heed that he put not his head between the

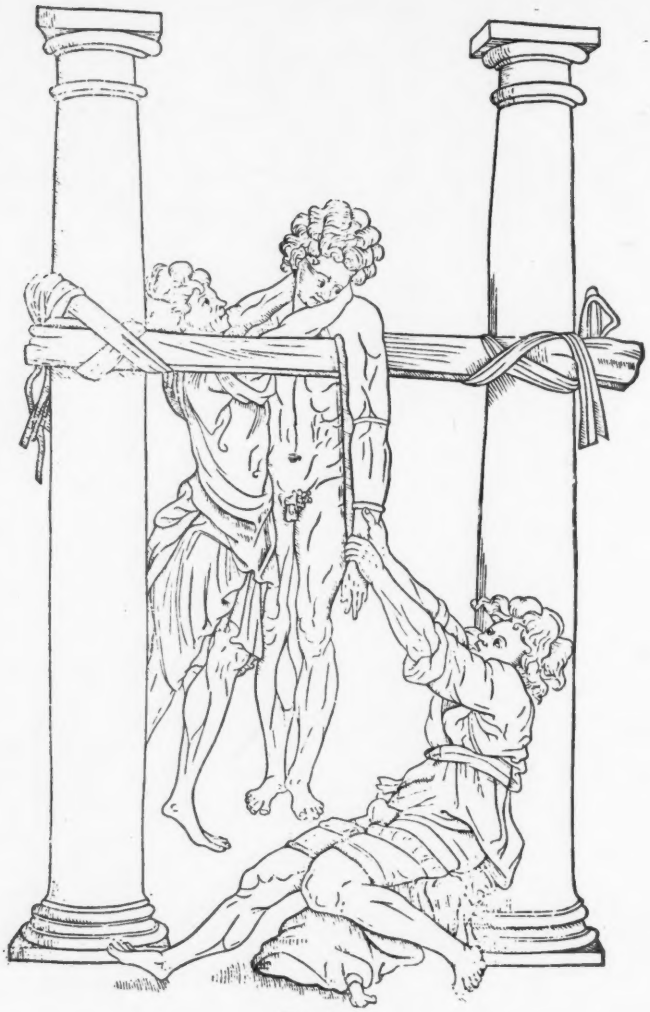

FIG. 7

"The Very Best Way of Restoring a Shoulder"; reduction over a beam. A door could be used. Note the wooden splint under the medial side of the arm. (From Vidius, *loc. cit.*) This was reputed to be the favourite method of Hippocrates.

steps. Then his arm, bound above the elbow with filletting or some other ligature fit for that purpose, shall be drawn down by the hand of some that assist you, and at the same time let the stool be plucked from under his feet so that he may hang upon the ladder. Thus by this means the head of the shoulder will be restored by itself, the endeavour of the surgeon assisting and pressing down on the shoulder blade, and moving it to and again. The bone being set, the stool which a little before was plucked from under the patient's feet shall be put there again that he may with the more ease and less pain, pull back his arm from the step of the ladder; for if he should lift it high up to draw it over, there would be danger, lest being newly set, and not well staid, the head of the bone might fall out again. I have

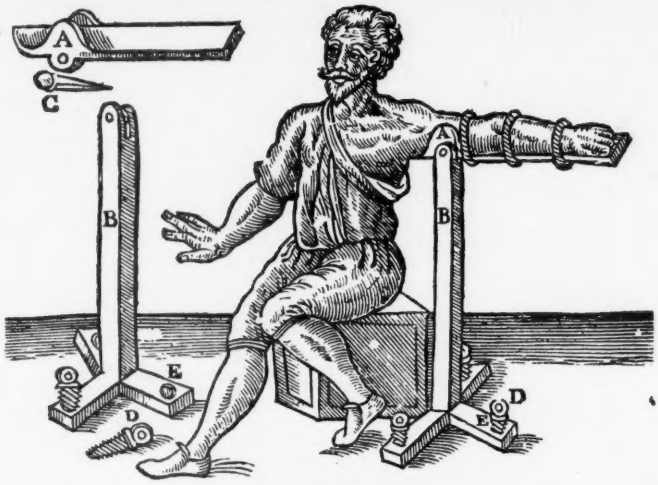

FIG. 8

An alternative method (Paré—*loc. cit.*). The use of a hinge and of a shaped splint is fully explained by the illustration.

FIG. 9

Reduction of the dislocation by a mediaeval type of screw-traction. This picture, from Scultetus (1693) is not much later than Paré and represents a technique which does not appear to have been known to the Greeks. Scultetus (Johann Schultes, 1595–1645) was famed for his illustrations of surgical instruments and appliances.

FIG. 10

The use of the rack to reduce a shoulder dislocation. (Vidius—*loc. cit*)

thought good to have all these things here expressed, that you may learn this operation as if you see it done before you.

" If you have never a ladder you may use a piece of wood laid across two posts. Also you may use a door as the other figure shows wherein you must observe a flat piece of wood with strings thereat." (Figs. 5–6.)

" Of the sixth manner of restoring a shoulder luxated into the arm-pit "

" Hippocrates writes that this is the best way to reduce a dislocated shoulder. You must take a wooden spatula some four or five fingers' breadth and some two fingers' thickness or less and some yards long. The upper part must be wrapped about with a linen or woollen rag that it may be the softer and hurt the less and then it must be so thrust under the arm-pit that it may thoroughly penetrate into the inner part between the ribs and the head of the shoulder bone, and bound by the strings to the arm. Next you must make use of a ladder, door, bedposts, or such like thing as shall be present at such a height that the patient may stand on tip-toes. Now this is the very best way of restoring a shoulder." (Fig. 7.) Fig. 8 illustrates an alternative and rather more complicated method mentioned by Paré.

The last two pictures accompanying this paper are not concerned with Paré's text. Fig. 9 which appears in Scultetus (Armamentarium Chirurgicum, 1693) is reproduced largely to demonstrate that screw-traction is much older than its present-day users, and Fig. 10 (from Vidius—Chirurgia, 1544) shows the rack, the classical mediaeval instrument of torture, being used as a means of reduction of a difficult shoulder dislocation, the usual role of the appliance being in this case reversed.

Very little need be said about these pictures but it is interesting to note that backward dislocation was not recognised in 1600. It is also of interest to observe that although some little advance has been made since the days of Hippocrates, yet most of the manoeuvres described are frankly plagiarised from the Greek. The text which is quoted is, in places, almost a translation of parts of the writing of Hippocrates (Worthington 1927).

Reduction of the shoulder around the operator's heel (Fig. 2) has long been styled the " Hippocratic method." It seems, however, that reduction over the operator's shoulder was actually the favourite method in the Greek gymnasia (Fig. 3). The addition of the weight of a child to the traction force is not seen in the one Greek illustration of the method which is available.

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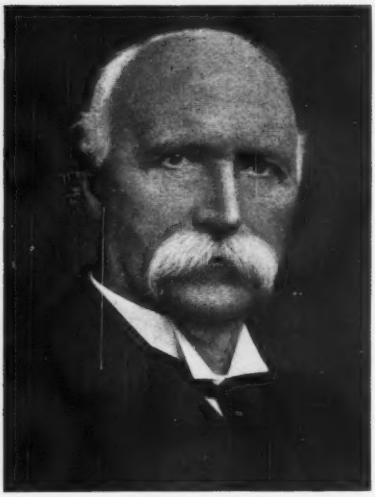
SIR WILLIAM MACEWEN (1848-1924)

An Eponymic Centennial Note

A century has gone full circle since Sir William Macewen was born on June 22, 1848, and yet his huge personality continues to overpower our generation. In the history of bone and joint surgery his name will forever be eponymously honoured through his operation which marks but one aspect of his life-long interest in bone problems. His classic monograph, "Osteotomy with an Inquiry into the Aetiology and Pathology of Knock-Knee, Bow-Leg, and other Osseous Deformities of the Lower Limbs," published in 1880 at the early age of thirty-two, is a succinct, lucid, comprehensive presentation of the subject, with the accent of finality on every page. Translated into many languages, it spread his fame throughout the civilised world. This extract is characteristic of the style:—



"On April 11th, 1875, I performed antiseptic osteotomy on a girl, six years of age, who had osseous anchylosis of the right knee, following suppuration of the joint, rising as a sequela of scarlet fever. The limb was straightened, and the wound healed as a typically antiseptic wound does. This was the first case of osteotomy performed in Britain. The second was performed by Lister during the winter session 1875–76. . . . Antiseptic osteotomy was performed for the first time in London by Mr Barwell (Richard Barwell, F.R.C.S., Surgeon to Charing Cross Hospital) on December 2nd, 1876. During the last few years antiseptic osteotomy has been greatly extended, and it has met with such success as to ensure it a permanent place as a safe, certain, and most successful procedure." (p. 62.)

The following extracts may be quoted from his "Clinical Lecture on Antiseptic Osteotomy: Having Special Reference to the Kind of Instruments Used, and to the Surgical Anatomy of the Extra-Articular Operation by Simple Incision at the Lower End of the Femur for Genu Valgum, delivered at the Glasgow Royal Infirmary." [1]

"In this morning's remarks references will be made, among other points, to the following: the age suitable for such operations; the kind of instruments employed, and how to use them; and the surgical anatomy of the site selected for my operation of simple femoral incision in genu valgum. . . . The chisel ought to be made to suit the material upon which it is to act. . . . It ought to be made of very fine steel, tempered so as to prevent brittleness or too great softness. . . . To employ a single word which would at once distinguish it from the ordinary chisel and at the same time be more euphemistic to a patient's ear, it might be called an *osteotome*."

The first set of osteotomes was made in 1876 by T. H. MacDonald, a blacksmith of Glasgow, under Macewen's direct supervision. The instrument was forged in a single piece and highly polished, at a time when bone-handled scalpels and wooden-handled chisels were universally employed, for Macewen had clearly grasped the principles of aseptic surgery.

Sir William Macewen's was a magnificent figure, with chiselled features and piercing grey eyes. His voice was low and mellow and, even when lecturing, hardly raised. He had an intense dislike to be photographed. A difficult character, he was essentially an individualist, unwilling or unable to share his ideas with colleagues or juniors; and he failed to win the affection of those who were his most devoted disciples.

W. R. BETT.

[1] British Medical Journal, 1879, **1**, 656–658.

SIR H. A. THOMAS FAIRBANK, D.S.O., M.S., F.R.C.S., M.Ch.Orth. (Hon.)

" Haec studia adulescentiam acuunt, senectutem oblectant "

Sir Thomas Fairbank may be the oldest member of the British Orthopaedic Association, but in mental vigour he is one of the youngest. Regarded with affectionate respect, he is sought by young and old for his encyclopaedic knowledge and sound clinical judgment which are based upon a vast store of remembered experience. He has an insatiable thirst for knowledge and in the words of Cicero " the studies which were an impetus to his youth do now in age provide his delight."

In Fairbank we see embodied much of the history of the British Orthopaedic Association. Orthopaedics in Great Britain arose from two main stems: from Liverpool through Hugh Owen Thomas and Robert Jones; and from London through Jackson Clarke, Tubby, Muirhead Little, and Openshaw, who were general surgeons. Of the next generation—Elmslie, Trethowen, Bankart, Fairbank and others—Fairbank was first to gain appointment as orthopaedic surgeon to a London teaching hospital, the Charing Cross Hospital in 1906. Similar appointments were soon established elsewhere, but for the first thirty years of this century London orthopaedics grew despite opposition and obstruction, and it was the sincerity and integrity of this modest man which counted for so much in gaining the co-operation of general surgical colleagues.

Through family influence, Fairbank intended originally to go in for dentistry. He qualified in medicine but also took the dental diploma. Indeed, if it had not been for the South African War, he might well have become established as a West End surgeon-dentist. But he served as medical officer with the troops in South Africa, and on his return was appointed Medical Superintendent at the Hospital for Sick Children, Great Ormond Street.

He saw the demonstration by Lorenz in January 1903 at the City Orthopaedic Hospital and three weeks later, being then a clinical assistant, he reduced his first congenital hip dislocation. Here also he came under the influence of Lane whose teaching of the " non-touch technique " he accepted and thereafter never ceased to teach and practise. It is said that in the twenty-eight years he worked at Great Ormond Street, and did much of his most important work, he was not communicative; but he taught by example, by the skilful handling of children, the careful management of orthopaedic appliances, and by the qualities of neatness and attention to detail. Sir Thomas has generally managed through gentle but firm persuasion to get his own way, and this was illustrated in his successful breaking of a long tradition of the London hospitals by which all out-patients were seen by assistant surgeons regardless of the fact that they had been treated in the ward by senior surgeons who never saw them again. Throughout the time that Fairbank served as senior surgeon, from 1913 to 1928, he shared with his assistant both in-patient beds and out-patient follow-up clinics. This practice, started at Great Ormond Street, is now almost universal throughout Great Britain.

Before the 1914–18 War, Fairbank toured North America and gained stimulus from Royal Whitman, Lovett, and Abbott, and from Goldthwait whom he had met years before in Liverpool with Robert Jones. He was a keen territorial soldier and, when war broke out, was soon in Flanders with the 85th Field Ambulance. It was only by the folly of military regulations that this mature surgeon continued to serve as junior medical officer while young men he had trained were given first-class surgical appointments. In due course, however, this was remedied. He became surgical consultant to the Army, taught bone surgery to men who were destined to become orthopaedic leaders of the future, helped to establish in the

Balkans a unit for the segregation and rehabilitation of injured men, served in the first Orthopaedic Centre at Shepherd's Bush, and took a prominent part in establishing the principles of treatment which are now generally acknowledged.

On returning to Great Ormond Street he restricted his practice to orthopaedics, and shortly afterwards was invited to establish a new orthopaedic department at King's College Hospital. Thus started a new and fruitful phase, loyally supported by general surgical colleagues and assisted at King's by Dudley Buxton who, in 1922, helped him to establish the first fracture clinic in London for ambulatory cases. At that time he was joined by Sister Bradbury from Great Ormond Street, a devoted assistant who has done so much to maintain

Sir Thomas at his desk.

his unique collection of radiographic and clinical material. In 1926, on the suggestion of Sir Henry Gauvain, he was appointed as first consulting orthopaedic surgeon to the Lord Mayor Treloar Hospital at Alton. This was the last hospital appointment he gave up, in 1946.

No record of the contribution of Sir Thomas Fairbank would be complete without comment upon his phenomenal work during the 1939–45 War. The Minister of Health invited him to act as chief orthopaedic adviser for the Emergency Medical Service. His responsibilities included expansion of existing orthopaedic facilities, development of facilities which did not exist, co-ordination of the work of regional advisers, negotiations with Service departments, and discrimination in the demand for trained orthopaedic surgeons between the needs of civilians on the home front and the expanding requirements of the armed forces. His labours were crowned with a degree of success which none other could have achieved, for he was held in such high respect; it was known that being armed with the powers of a dictator he seldom used them; and it was recognised that the force he preferred was that of

reason and persuasion. Those who know him at the periphery are still astounded by his memory of patients, and knowledge of the details of local hospital organisation.

It is in the clinic that Sir Thomas' discernment and knowledge is so striking. On one occasion at Great Ormond Street, on hearing the peculiar and piercing scream of an infant at the other end of the out-patient hall he remarked: " That sounds like the cry of infantile scurvy." And so it was. He is a careful operator, always conscious of the patient; no trouble is too great and there is no excuse in his vocabulary. His teaching is intimate, helpful, and amusing.

Fairbank's contribution to orthopaedic literature is voluminous. He speaks often at medical meetings and always with something worth saying. The period of most prolific writing dated from his appointment to King's College Hospital after the first war, but already he had made important observations on the treatment of Erb's palsy, the correction of con-

The old Hospital for Sick Children, Great Ormond Street.

tracture of the subscapularis for which his operation was devised, and the treatment of congenital dislocation of the hip joint which he discussed with increasing knowledge and experience in later years. In 1922 he was able to report upon one hundred and seventy-five congenital dislocations treated between 1903 and 1914. The Lady Jones Lecture, published by Fairbank in 1930, included a fascinating discussion of the pathological anatomy of congenital hip dislocation, the mechanism of limp, the ligamentous and muscular slings which support the pelvis on the femur, the power of adduction of these muscles, and the part they play in the Trendelenburg gait. It can still be read with profit by all who deal with these dislocations.

A few weeks ago Fairbank put his views as follows:—" Congenital hips are of three types: 1) imperfect acetabulum, with the femoral head in the joint, but slightly eccentric; 2) subluxation, including ' marginal displacement '; 3) dislocation on the dorsum ilii. The first is commonly overlooked until adult life when arthritis develops. It may be seen in the opposite hip, previously regarded as normal, after a unilateral dislocation has been dealt with. If spotted early enough operation to improve the acetabulum is well worth consideration. In the second type when the head rests exactly opposite the upper limit of the socket, the

prognosis is very bad unless an operation is performed to improve the roof of the acetabulum and make a lip. If the child is only two years of age or less, I think it is worth trying conservative measures, but if approaching the age of four I think it is essential, after manipulation to get the head central and the limb in a stable position, to follow this by the so-called upper lip operation.

" Open reduction *per se*, without an upper lip operation, has no advantage over closed reduction, since ultimate success always depends upon retaining the limb in the chosen stable position long enough to allow the acetabulum to acquire a shape that is approximately normal. I am not maintaining that open reduction is never advisable, for I think it occasionally is; for example when manipulation fails to achieve reduction, the child being sufficiently young. The hip should also be opened if after manipulation it is found to be exceptionally unstable, or the X-ray shows the head is not really home in the acetabulum.

" In the young, under three years of age, a subluxated and a luxated hip should be treated in the same way, namely by manipulative reduction (simple gradual abduction if under twelve months) and prolonged fixation. I believe that further manipulations in various directions, carried out after reduction and before applying the plaster, are of real value in getting the head really home and getting rid of possible folds of capsule nipped between the bones. I think it is a mistake to include the knee in plaster as a routine, because rotatory movement of the head in the acetabulum is useful, and one likes to get the hamstrings stretched. Ante-version I believe seldom calls for correction.

" My forty-five years experience of this problem leads me to emphasise the difficulty of what I would call the ' diagnosis of prognosis.' Recently reviewing some of my twenty-year results I have been struck by the very good results obtained in cases that at first had seemed imperfect, and vice versa. The one criterion I would stake everything on is the centering of the femoral head in the acetabulum."

In making a life-time study of the complexities and problems of general pathological lesions of the skeleton, Sir Thomas has done much to bring order out of chaos. His Presidential Address to the British Orthopaedic Association (British Journal of Surgery 1927) is a classic of modern orthopaedic literature, in which is classified and distinguished a great variety of developmental errors in bone formation, disturbances of bone structure, disorders of metabolism, and deviations of growth. He returned to the subject in the first Robert Jones Memorial Lecture (British Journal of Surgery 1939), discussing skeletal lesions with increased and decreased density of bone, and emphasising the part played by fibrosis of the marrow. More recently, under the title of " Dysplasia Epiphysealis Multiplex," he presented to the Royal Society of Medicine a critical analysis of disturbances of epiphyseal growth, isolating a group of cases with mottled bone formation differing from the changes of Perthes' disease, dyschondroplasia, dysplasia epiphysealis punctata, Morquio-Brailsford anomaly, etc.

Sir Thomas is a collector of the rarities of bone pathology. But he is more than a collector; by endless thought he is handing back his records, and those referred to him by many friends and colleagues, to the common pool of well-ordered classification. With an unrivalled experience of congenital dislocation of the hip joint his great delight is to discuss new ideas with junior members of the profession who have studied individual cases in detail. Of congenital talipes equino-varus he believes that " failure to get good results is due entirely to the failure of surgeons to take infinite pains with every case; this ultimately is the secret of the Denis Browne method, which in other respects is a good method." One of his greatest recent pleasures was to see the precision, attention to detail, and timeless consideration with which Smith-Petersen performed an arthroplasty of the hip joint in London. Of the " clever surgeon " who is technically dexterous, Fairbank comments: " Of far greater importance is the man's intellectual integrity."

" Great men are the guide-posts and landmarks in the State " said Edmund Burke. Of such is Thomas Fairbank. N. C.

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PROCEEDINGS AND REPORTS OF UNIVERSITIES, COLLEGES, COUNCILS AND ASSOCIATIONS

CANADA

JOINT MEETING OF THE CANADIAN, BRITISH AND AMERICAN ORTHOPAEDIC ASSOCIATIONS

Canada is to serve as host for the combined meeting of the American, British and Canadian Orthopaedic Associations which will be held in Quebec City in June. The American Orthopaedic Association will be led by their president, Dr Robert I. Harris of Toronto. Dr J. Edouard Samson of Montreal will preside over the meetings of the Canadian Orthopaedic Association. The British Orthopaedic Association will be represented by the president, Mr Alan S. Malkin of Nottingham,* and a small group of surgeons

The Joint Meeting of Canadian, British and American Orthopaedic Associations will be held in June 1948 in Quebec, the oldest city in Canada, and the centre of Government of French Canada. This photograph, reproduced by courtesy of Canada House, London, shows the Provincial Parliament Buildings of the City.

who will endeavour to make good their lack of numerical strength by the vigour of their youth. The programme has been arranged by a committee under the capable chairmanship of Dr John L. McDonald of Toronto, whose organising ability was once demonstrated in Chicago when he saved the life of the British Editor of this Journal by disguising him as "Mr Smith." Clinical presentations, symposia, short

* The Editor would take this opportunity of regretting that in the first British number of the Journal the name of Mr Alan S. Malkin, who was then president-elect of the British Orthopaedic Association, was not included in the list of representatives of the British and American Orthopaedic Associations who joined in the London Conference on future developments of the Journal of Bone and Joint Surgery. Having regard to the important part he played, the omission was all the more unfortunate, but we know that our respected president will forgive the oversight.

papers, executive sessions, and banquets, will occupy every moment from early Thursday morning, June 3, until late Sunday evening, June 6. The provisional programme includes these papers and discussions:

Aseptic Necrosis of Bone and Fractures of the Neck of the Femur—Edward Compere, Chicago; W. W. Plummer, Buffalo; Dallas Phemister, Chicago; James Patrick, Glasgow; Jackson Burrows, London; W. Edward Gallie, Toronto.

Subtrochanteric Leg Shortening—Lawson Thornton, Atlanta; Discussion—Walter P. Blount, Milwaukee.

Ischio-femoral Arthrodesis of the Hip Joint—Herbert A. Brittain, Norwich; Joseph Freiberg, Cincinatti.

Osteoid Osteoma—Malcolm Dockerty, Rochester; Discussion by Ralph Ghormley, Rochester; Campbell Thomson, New York.

Slipping of the Upper Femoral Epiphysis—Beckett Howorth, New York; Discussion by S. Kleinberg, New York; Armin Klein, Boston.

Arthroplasty of the Hip Joint—Alexander Gibson, Winnipeg; J. S. Batchelor, London; Discussion by Paul C. Colonna, Philadelphia.

Arthroplasty of the Knee Joint—J. S. Speed, Memphis; Philip C. Trout, Roanoke; J. Edouard Samson, Montreal; Discussion by Reginald Watson-Jones, London.

Changes in Elastic Adipose Tissue with Advancing Years—J. G. Kuhns, Boston; A. W. Farmer, Toronto.

Physiological Blocking of Flail Joints—Alberto Inclan, Havana; Discussion—Fremont Chandler, Chicago.

Fracture-dislocation of the Pelvis—Frank W. Holdsworth, Sheffield; H. Earle Conwell, Alabama.

Developmental Coxa Vara—A. B. Le Mesurier, Toronto; Discussion by W. T. Green, Boston.

Congenital Dislocation of the Hip Joint—Juan Farill, Mexico; Fred C. Durbin, Exeter; Discussion by Henry R. McCarroll, St Louis.

Arthrodesis of the Ankle Joint—W. Edward Gallie, Toronto.

Oestrogens and Bone Formation in the Human Female—Mary S. Sherman, Chicago; C. Howard Fletcher, Chicago, Fuller Allbright, Boston; Arthur Ham, Toronto.

Congenital Discoid Meniscus; Results of Menisectomy; Knee Joint Changes after Menisectomy—Ian S. Smillie, Edinburgh; John C. Charnley, Manchester; T. J. Fairbank, London; Joseph S. Barr, Boston.

Cervical Fractures and Fracture-dislocations—William A. Rogers, Boston; W. G. Turner, Montreal; William Cone, Montreal; E. A. Nicoll, Mansfield, Nottingham.

Organisation of an Accident Service—W. Gissane, Birmingham, England; J. Heberling, Pittsburgh.

Presidential Address—Robert I. Harris, Toronto.

Undergraduate Teaching in Orthopaedic Surgery—Bruce Gill, Philadelphia; George Perkins, London.

Operative Approaches to the Shoulder Joint—Le Roy Abbott, San Francisco; George Bennett, Baltimore.

External Fixation in Orthopaedic Surgery—J. R. Naden, Vancouver; Discussion by E. C. Janes, Hamilton; John R. Moore, Philadelphia.

Fractures of the Shaft of the Femur—E. Harlan Wilson, Columbus; John C. Charnley, Manchester.

Denervation of the Elbow Joint for the Relief of Pain—James E. Batemen, Toronto; J. Albert Key, St Louis.

Pedicled Nerve Graft—F. G. St Clair Strange, Folkstone, England.

Elephantiasis with Congenital Bands in Children—A. W. Farmer, Toronto; H. B. Macey, Temple, Texas.

Scoliosis with Paraplegia—K. G. McKenzie, Toronto; F. P. Dewar, Toronto; Arthur Steindler, Iowa City.

Paper by Sten Friberg, Stockholm, Sweden.

Nuffield Fellows and Travelling Fellows in Orthopaedic Surgery—The appointment of ten Nuffield Fellows, and two Travelling Fellows in Orthopaedic Surgery was recorded in the last issue of this Journal. Mr James Wishart, first assistant at the Robert Jones and Agnes Hunt Orthopaedic Hospital, has been appointed a third Travelling Fellow. All these surgeons are to be met on arrival in New York by the president of the American Orthopaedic Association. Thereafter they will be received in the clinics of New York, Boston, Philadelphia, Baltimore, Washington, Quebec, Montreal, Toronto, Detroit, Ann Arbor, Chicago, St Louis, and Rochester. A six weeks' tour of American and Canadian orthopaedic centres has thus been planned; travelling has been facilitated; generous hospitality has been arranged; and expenses are being defrayed by American and Canadian friends of Britain. We trust only that these young British surgeons will seal once more, in their own generation, the bonds of friendship which already exist in a former generation.

GREAT BRITAIN

ORTHOPAEDIC SECTION OF THE ROYAL SOCIETY OF MEDICINE

A well-attended meeting of the Orthopaedic Section of the Royal Society of Medicine was held in the late summer, 1947, under the presidency of Mr V. H. Ellis, at the Lord Mayor Treloar Hospital, Alton, when a clinical demonstration was arranged by the medical superintendent, Mr Stanley Evans. Cases of Brittain's ischio-femoral arthrodesis were demonstrated by Mr Langston. Professor T. P. Kilner read a paper on Plastic Surgery in Relation to Orthopaedic Surgery.

Contributions of the late Sir Henry Gauvain to Orthopaedic Surgery

Sir Thomas Fairbank said that when Gauvain was appointed to take charge of the hospital in 1908 there were definite signs of a swing from the radical to the conservative treatment of non-pulmonary tuberculosis. In the same year, one of Gauvain's teachers, the late Sir Anthony Bowlby, published a paper on tuberculosis of the hip joint, based on 900 cases under twelve years of age, treated at the Alexandra Hospital. He condemned operative treatment which at that time was almost universally adopted, and showed that better results could be achieved by conservative methods. Gauvain was an enthusiastic advocate of these methods. The first of many papers was written in 1910 in collaboration with Dr Calvé, and dealt with the treatment of tuberculous abscesses by aspiration instead of incision. Many of his most valuable publications dealt with general or adjuvent treatment, in which he did pioneer work. He supported the idea that tuberculous arthritis was no more than a local manifestation of generalised infection. Heliotherapy had been adopted earlier in the century by Bernhard, and by Rollier, but quite independently Gauvain was convinced of the value of sunbathing. He often paid tribute to the value of this method of increasing the powers of resistance of the patient. He insisted on the need for graduation of exposure, and insisted that exposure in the shade was no less necessary than exposure to the sun. He stressed the value of alternation of stimuli. Sun treatment was allowed only under the supervision of a specially trained sister. He was convinced of the psychological value of this treatment.

Grand Terrace, Lord Mayor Treolar Cripples' Training College and Hospital; and the late Sir Henry Gauvain, Medical Superintendent, 1908–1945.

Open-air treatment was carried out in all weathers. Believing that sea-bathing and a change of climate would benefit selected cases, he persuaded the Trustees to open a branch at Hayling Island. He found, however, that while most cases responded well, some were affected adversely, and constant care was necessary. Sea-bathing was considered valuable even for recumbent cases, but elaborate precautions were taken to avoid chill by the use of windscreens, braziers, and hot meals. With the support of the Medical Research Council, he collaborated with Sir Leonard Hill and Dr Argyll Campbell in carrying out metabolic researches which showed that exposure to air had a much greater influence than exposure to sun, and proved that the two together might increase the metabolic rate by as much as 40 per cent.

Gauvain took a leading part in the introduction of celluloid splints for the later treatment of tuberculous cases. The celluloid solution was rendered non-inflammable by mixing with calcium chloride. He was impressed by the work of Finsen, and by the use in Denmark of artificial light. He went to Copenhagen to study their methods. It was not until after the First World War that he was able to open a Light Department, fully equipped with all the latest apparatus, and with a sister in charge who had been sent to Denmark for special training.

He was a pioneer in the development of hospital schools, insisting on the importance of discipline and full occupation of the time of the patient with work and play. Occupational therapy had been a feature

at the Treloar Hospital for many years, especially among older girls in whom it served both as diversion and as training for later employment. He also started one of the first crippled boys' colleges where they were taught to earn a living. To sum up, Gauvain took a leading part in introducing all the features which now, as a matter of course, were included in the modern country hospital.

Sir Thomas Fairbank said that in one paper, published in the Lancet, November 16, 1918, Gauvain described a method of testing for pathological activity or quiescence of arthritis in tuberculous hip disease. If the condyles of the femur were grasped by one hand, the head of the bone could be rotated in the acetabulum. When this movement was checked, further sharp rotation was followed instantly, when disease was active, by protective spasmodic contraction not confined to the muscles about the joint, but extending to the abdomen where it was felt easily by the palm of the other hand placed above the pubes. Gauvain also pointed out that if disease was not quiescent this rotation of the femur with slight force was transmitted to the pelvis and the iliac spines.

" I cannot conclude these remarks " said Sir Thomas " without referring to the exceptional charm of Henry Gauvain's personality, which endeared him so much to all with whom he worked. Henry Gauvain made this hospital: he *was* the hospital. The happy and optimistic atmosphere that he engendered, and which was evident in every member of the staff, infected the patients and played an important part in their progress and recovery."

Orthopaedic Surgery in Rheumatoid Arthritis—*Mr Alexander Law* (London Hospital) on January 14, 1948, emphasised the importance of undertaking reconstructive surgery before joint destruction was complete and before serious atrophy of muscles and ligaments had developed. He described the indications for excision of the acromion process in rheumatoid arthritis of the shoulder joint; for excision of the head of the radius with synovectomy in certain cases of arthritis of the elbow; for complete arthroplasty of the elbow joint; for excision of the lower end of the ulna by which to restore radio-ulnar and wrist movement; and for arthroplasty of the hip joint by the Smith-Petersen vitallium-mould technique. He emphasized the necessity for shaping congruous joint surfaces by means of special instruments, and for creating a large acetabulum; and said that success depended not only upon careful and experienced operative technique, and an intensive post-operative regime, but upon willingness to repeat operative procedures a second or even a third time on the same joint of the same patient. There was no short cut to success. It was pointed out that a satisfactory arthroplasty of the knee joint had not yet been developed but that recent work showed promise. In the treatment of fixed kyphotic deformities of the spine, especially in spondylitis ankylopoetica, spinal osteotomy by resection of the posterior neural arch and facets was worth undertaking.

Serious Limitations and Erroneous Indications of Biopsy in the Diagnosis of Bone Tumours—At the meeting held on January 16, *Dr James Brailsford* urged that punch or drill biopsy of bone tumours was unreliable, and that even open biopsy was misleading and led to serious error. He suggested that there might be radiographic evidence that biopsy caused general dissemination. Radiographic diagnosis, particularly when based on serial examinations, was considered to be much more reliable. In the course of subsequent discussion Dr Brailsford's dogmatic views were found to be generally unacceptable. *Dr Frank Ellis* pointed out that radiologists varied in their competence no less than pathologists. Serious misinterpretation might arise from radiographic examination just as readily as from pathological examination. He believed in fact that errors after biopsy were infrequent. *Mr W. D. Coltart* said that diagnosis must always be based on the correlation of clinical, radiographic, and pathological evidence.

Curare (Intercostrin) in the Treatment of Acute Anterior Poliomyelitis—A film by *Dr Nicholas S. Ransohoff* (New York), which had been brought to this country by Sir Morton Smart, was introduced by Sir Reginald Watson-Jones at the meeting held on February 3, 1948. Dr Ransohoff accepted the evidence of Plato-Schwartz that in acute anterior poliomyelitis there was widespread muscle spasm, and believed that such spasm was responsible for dysphagia, respiratory embarrassment and early onset of deformity. By injecting curare the spasm was relieved, Sherrington's law of reciprocal innervation was re-established, and the pathological stretch reflex was obliterated. The dosage employed was 0·9 units per kilo of body-weight every eight hours for the first twenty-four hours; the dosage thereafter being increased, if there was no adverse reaction, to 1·5 units per kilo of body-weight every eight hours. Intra-muscular injection was continued until all evidence of muscle spasm had disappeared.

The essential principle of Ransohoff's treatment was not simply to give curare injections, but to make use of such injections in order to institute passive stretching, exercise, weight-bearing, and ambulation at the earliest possible moment. The exercises and movements were started within twenty-four hours of admission. At three separate intervals during the first three hours after each day-time dose of curare, every joint of the body was put through as complete a range of movement as possible. All muscles were stretched in the attempt to re-establish normal muscle length. The patient was put on his feet as early as possible, no attempt being made to avoid fatigue. Throughout the hours that joints and muscles were not being stretched the patient was made to exercise with parallel bars, specially constructed chairs, trapeze, mariner's wheels, bicycles, and every type of apparatus. The patients were always busy.

It was emphasised that no claim was made to have established a cure for anterior poliomyelitis, but that this treatment gave better results more quickly than any other method. In a series of twenty-nine patients, no fixed deformities or contractures developed. Five patients with bulbar paralysis responded in a surprising manner to adequate doses of curare, the relief being attributed to relaxation of spasm of the accessory muscles of respiration and the diaphragm. Dysphagia responded immediately.

Sir Reginald Watson-Jones (London) said that although he had introduced the film it must not be thought that he accepted Ransohoff's principles. He quoted the words of Voltaire: "I utterly disagree with what you say, but I would defend to the death your right to say it." He thought that further electromyographic investigation was needed into the question of spasm in poliomyelitis, and the extent to which spasm was an important factor calling for special treatment. Further evidence was needed before it could be accepted that treatment by the injection of curare altered the prognosis and improved the outlook. That part of the treatment which insisted upon early mobilisation and weight-bearing, passive stretching despite pain, and exercise despite fatigue, was in startling contrast to the principles advocated by Hugh Owen Thomas and Robert Jones. It might be that in the past we had emphasized too much the importance of protecting paralysed muscles. It might be that avoidable joint stiffness had sometimes been allowed to develop. It was possible that earlier mobilisation and ambulation was advisable. But he would need much more than the evidence of this film to persuade him that fatigue and exhaustion of weak and partly paralysed muscles was desirable. Still more did he feel revulsion, amounting almost to horror, when he saw painful joints and muscles being stretched and forced despite the agonised protests of the patient.

Professor H. J. Seddon (Oxford) agreed that deformity after poliomyelitis was probably due to the development of contractures rather than to muscle imbalance *per se*, though the latter was, of course, a source of deformity. If this notion was correct then maintenance of a full range of movement was of great importance. The treatment of poliomyelitis had been dominated in this country by the belief that paralysed or paretic skeletal muscle was in so delicate a state as to require prolonged rest. There was no longer any doubt that denervated muscle did not degenerate as did, say, a divided nerve. It merely atrophied; and putting a paralysed muscle through its full range was no more harmful than stretching a normal muscle, though maintenance of a muscle in an overstretched position was clearly harmful whether the muscle was denervated or not.

If it was accepted that preservation of mobility was necessary, we were faced with the difficulty of how to achieve it despite limitation by pain. Dr Ransohoff believed that pain was due to muscle spasm and that if spasm could be abolished the pain would also be relieved. But there was no clear evidence that a motor neurone disturbance causing muscle spasm also caused pain. In spastic paralysis painful limitation of movement did not occur except in late neglected cases where contractures had been allowed to develop. It was much more likely that pain was the cause of spasm. Unfortunately, we knew very little about the cause of painful phenomena in poliomyelitis, except that extensive changes in the posterior root ganglia had been found with some frequency in fatal cases as well as in experimental disease. Muscle spasm, though by no means a constant feature of the disease, was very real and could be demonstrated electromyographically; it might be independent of the pain or it might be caused by it. In either case it was difficult to see how curare, acting on the motor end-plates, could relieve it. Did it, in fact, do so?

Professor Seddon said that the cases he had seen in Dr Ransohoff's clinic in New Jersey had impressed him most favourably, but it so happened that at that time all were past the acute phase of the disease and none was receiving curare. He had been told by other surgeons who had seen manipulations after the administration of curare that patients suffered very considerable pain. The agonised expressions shown in this film were disturbing. He could not help questioning the efficacy of the drug as a remedy for painful limitation of movement in poliomyelitis, and believed that the treatment derived its success more from the determination of physiotherapists than from the action of the drug.

There was now little doubt that in poliomyelitis there was disorder of reciprocal innervation; the electromyographic evidence was convincing. Unfortunately, in this country, we had not been able to assemble the apparatus in time to make observations during the recent epidemic. Dr Ransohoff claimed that curare restored the normal reciprocal activity of antagonistic muscles, but unless the drug had some fairly powerful central action it was impossible to see how it could act in this way.

Dr Ransohoff's second argument was that patients should be got out of bed as early as possible; if they were bedridden for many months they forgot how to walk. At Oxford, during the recent epidemic, patients had been allowed up much earlier than had formerly been considered safe, but with nothing like the rapidity favoured by Dr Ransohoff. In every case a careful watch had been kept for deterioration in muscle power. Slight decrease in power had sometimes been observed in such muscles as the abductors of the hip. Yet in these cases slackening of the pace arrested the deterioration, and in almost all cases there had been gradual increase in power whether weight-bearing was allowed or not. This was hardly the occasion for discussing the rather complicated phenomena of recovery from paralysis in poliomyelitis,

but he thought that Dr Ransohoff's arguments in favour of early ambulation were much nearer the mark than the slavish enforcement of bed-rest that most of us had practised.

Dr Ransohoff's last point, that very early active exercises should be instituted, was altogether unsound. If recent experience in the treatment of meningitis was any guide, then the presence of protein in the cerebro-spinal fluid was an indication of persistence of active inflammatory changes in the cord and meninges. In poliomyelitis, protein might be found as late as eight weeks after the onset of the disease, and it hardly seemed rational to encourage activity in the anterior horn cells until after the acute process had subsided.

Clinical Meeting, Section of Orthopaedics, March 1948—Mr Nissen showed a case of *congenital bowing of the tibia* in which pathological fracture had been treated successfully by massive posterior bone grafting. Mr T. H. Meyer showed a most successful *arthroplasty of the knee joint* in a woman aged fifty-five years with active rheumatoid arthritis whose other knee had been arthrodesed. No tissue had been interposed between the bone ends. Care had been taken to remove the back of the femoral condyles. The result showed unusually good stability, and a range of movement from full extension to right-angled flexion. Mr H. L. C. Wood showed a case of *idiopathic hypertrophy of the mandible*. Mr George Bonney reported the result of treatment by immobilisation in plaster of *Sinding-Larsen-Johansson disease of the patellar apices*. One knee was immobilised and on that side the discrete fragments had fused and the symptoms were relieved. The other knee was left free; symptoms persisted and radiographs showed that the discrete ossicles had not fused with the main centre of ossification. A number of cases of *neoplasm of bone* were shown including a reticular-celled sarcoma of the humerus by Mr W. D. Coltart for which local excision was advised; a synovioma of the ankle joint by Mr Kessel; and various obscure tumours by Mr Hywel-Davies and Mr Robb.

Rupture of Intervertebral Discs in the Cervical Region

In the Section of Neurology of the Royal Society of Medicine, on March 4, 1948, *Dr Russell Brain* opened a discussion on rupture of the cervical intervertebral discs. He outlined all the causes of nerve root irritation due to lesions of the spinal intervertebral joints, and reviewed ten proved cases of cervical disc retropulsion.

Pathological Features—The frequency of retropulsion of a disc was greater in the cervical and lumbar regions than in the dorsal region by reason of the mobility, lordosis, and exposure to trauma. The earliest resulting changes were vascular in origin—the veins being affected first. The problem in the cervical spine differed from that in the lumbar spine not only because cervical roots were shorter, but because there might be involvement of the spinal cord as well as of nerve roots. Ten of Dr Brain's cases of ruptured cervical intervertebral discs had been verified at operation—nine by Mr Northfield and one by Sir Hugh Cairns: two had root signs alone; eight showed evidence of pressure on the spinal cord. Only in two patients had injury been a possible factor; one other occurred during pregnancy; and seven were associated with cervical osteoarthritis. In the traumatic group, congenital weakness of the disc might be a factor; one patient had previously suffered from sciatica. The group of cases with osteoarthritis were older; in these patients compression might arise partly or wholly from bone. The level involved was C.3–4 in one, C.4–5 in two, C.5–6 in six, and C.6–7 in one.

Clinical Types—Four clinical types could be distinguished: 1) acute onset of severe pain with root signs; 2) insidious onset of severe pain, sometimes intermittent, with root signs; 3) insidious onset with root and cord signs; 4) insidious onset with cord signs alone. Root pain was shooting or gnawing in character, aggravated by movement of the neck and upper limbs, and by coughing. There might be numbness or tingling in the corresponding dermatome. Sensory loss was patchy and limited in area. Reflexes might be diminished or lost. Fasciculation of muscles might be seen, but weakness and wasting were uncommon. Four patients showed the Brown-Séquard phenomenon, and in four there was bilateral cord pressure with greatest involvement of the pyramidal tracts and little involvement of the sensory tracts. There was abnormality of the cerebro-spinal fluid in seven of the eight cases, and myelography showed a filling defect in every case in which it was carried out.

Treatment—The essential treatment was immobilisation in bed with or without skull traction, or the use of a plaster or leather cervical collar. Operation should be reserved for cases with much wasting or with evidence of cord compression.

Review of thirty-seven cases—*Mr G. C. Knight* pointed out that pain seldom reached the distal limit of a dermatome. It might be referred over the front of the chest to the precordial area. Compression of the seventh cervical root caused severe precordial pain which was sometimes mistaken for cardiac angina. Pain was usually provoked by homolateral flexion of the neck, sometimes by extension, and not always by vertical compression. Electromyography might be useful in diagnosis. " The best treatment is a long waiting list," said Mr Knight. Manipulation was dangerous. Rest was advisable in the position of

greatest comfort—usually hetero-lateral flexion. Operation was indicated only when conservative treatment failed. If the nerve root only was involved, hemilaminectomy gave sufficient exposure; but if there was evidence of cord pressure, laminectomy and a transdural approach was advisable. Mid-line protrusions should not, if possible, be operated upon. He had records of thirty-seven patients, twenty-nine with root symptoms and signs, four with evidence of pressure on half the cord, and four with mid-line protrusions affecting both sides of the cord.

Review of forty-six cases—*Dr Aldren Turner* had studied forty-six cases, forty-five of which had been followed until all pain was relieved. He relied on bed-rest with the head supported with pillows. Relief came within three weeks. Paraesthesia lasted longer than pain. He stressed the importance in diagnosis of dysaesthesia of the index finger, pain on cervical movement, and signs of involvement of the seventh cervical root.

Anatomical Studies—*Mr John E. O'Connell and Dr H. Coombs* reported an anatomical study which showed that the lumbar intervertebral discs were twenty to twenty-five times as heavy as the cervical discs. He distinguished four clinical types of cervical disc lesion: 1) cervical and brachial pain, with symptoms and signs in the distribution of one cervical root, and with a good prognosis; 2) cord compression often coming on suddenly after vague cervical pain; 3) severe tetraplegia from compression of the cord after trauma; 4) cervical stiffness after injury with signs resembling those of amyotrophic lateral sclerosis.

Radiographic Studies—*Dr J. W. Bull* mentioned two anatomical characteristics of the cervical spine: 1) the relative smallness of the intervertebral discs; and 2) the presence of neuro-central joints. The joints between the skull and atlas, and between the atlas and axis, lay in front of the emerging nerve roots. The homologues of these joints in the rest of the cervical spine were the neuro-central joints which also lay in front, and the ordinary lateral joint between neural arches behind. The importance of directing the rays slightly upwards in taking antero-posterior radiographs of the cervical intervertebral spaces was emphasized. Oblique projections were necessary to visualise the lateral joints.

ROYAL COLLEGE OF SURGEONS OF ENGLAND

Elections ad eundem of Professor T. P. McMurray and Mr S. Alan S. Malkin—Mr T. P. McMurray, who recently retired from the chair of orthopaedic surgery in the University of Liverpool, and Mr S. Alan S. Malkin of Nottingham, president of the British Orthopaedic Association, both Fellows of the Royal College of Surgeons of Edinburgh, have been elected *ad eundem* to the Fellowship of the Royal College of Surgeons of England.

Lectures in the Royal College of Surgeons—Throughout April, May and June 1948, Lectures on Anatomy, Applied Physiology, and Pathology will be given daily in the College. On June 14, Mr Eric Lloyd will lecture on Fractures in Childhood, and on June 15, Sir Thomas Fairbank will lecture on Abnormalities of the Skeleton. The statutory and other lectures given during February and March included:

February	6	Repair of Tendon Injuries in the Hand—Hunterian Lecture—Professor R. G. Pulvertaft.
	6	Reactions of Bone to Injury—Professor S. L. Baker.
	12	Bone, Cartilage, and Fascia Transplants—Mr Rainsford Mowlem.
	16	Skin Flaps—Indications and Technique—Sir Harold Gillies.
	17	Treatment of Skeletal Tuberculosis—Hunterian Lecture—Professor M. C. Wilkinson.
	18	Free Skin Grafting—Methods and Application—Mr A. B. Wallace.
	18	Penicillin—Sir Alexander Fleming.
	20	Burns and their Early Treatment—Mr R. P. Osborne.
	25	Hand Injuries—Mr J. N. Barron.
	25	Actinomycosis—Mr Zachary Cope.
	26	Hand Deformities—Mr J. B. Cuthbert.
March	10	Manifestations of Boeck's Sarcoid—Erasmus Wilson Demonstration—Mr R. W. Raven.
	12	Recent Advances in the Treatment of Arm Amputations, Cineplastic Surgery, and Arm Prosthesis—Arris and Gale Lecture—Mr Leon Gillies.
	15	Acute Uraemia—Arris and Gale Lecture—Dr E. M. Darmady.
	22	Fractures of the Spine—Sir Reginald Watson-Jones.
	24	The Pectoral Girdle—Arnott Demonstration—Mr R. J. Last.
	25	The Knee Joint—Arnott Demonstration—Mr R. J. Last.

Flexor and Extensor Retinacula of the Wrist and Ankle—In a Hunterian Lecture, *Mr H. F. Lunn* said that the difference in function of the flexor and extensor retinacula depended on the fact that the flexor tendons, when in action, thrust against the underlying bone, whereas the extensor tendons thrust against the overlying retinacula. Consequently surgical division of the extensor retinacula might be expected to weaken the extensor power of ankle and wrist. The length of synovial sheath of a tendon

depended on a number of factors, of which the most important was change in the direction of the tendon. Hence the longest sheaths in the hand were those for the tendons of the most divergent digits. Another factor was the crossing of one tendon by another—for example the long sheath of tibialis posticus which was crossed by the flexor digitorum longus, and of the extensores carpi radialis which lay beneath the abductor pollicis longus and extensor pollicis brevis. Beneath the retinacula, the mesotendon with its contained blood-vessels was attached to that surface of the tendon least subjected to friction. Hence at the wrist the mesotendon was attached to the ulnar surface of the flexor pollicis longus tendon, but to the radial side of the flexor tendons of the fingers. The lecture was illustrated from dissections in the Anatomical Museum of the College some of which had been beautifully prepared by Dr D. H. Tompsett.

THE HUNTERIAN SOCIETY

It is customary for the Hunterian Society to celebrate each year, within the boundaries of the City of London, the day of birth of John Hunter—February 13 or possibly February 14. This year, austerity conditions made it necessary to substitute for the usual dinner a simple evening reception which was held in the West End, within a few hundred yards of St George's Hospital. Mr Roche, president of the Society, in receiving the guests and proposing the traditional toast, said that " if Hunter had lived to-day he would probably not have been born at all. He was the youngest of ten children and in these days families of ten are hard to find." Mr Roche wondered what might have been the reactions of John Hunter in attending a cocktail party in his honour 150 years after his death. Despite the temperance of his later years he might well have applied one of his favourite expressions, " Don't think; try it." In honour of the celebration, the Society displayed its treasures including Hunter's manuscripts, his walking stick (the handle of which was lost in the London blitz), his watches, balances, silver-rimmed spectacles, brass dispensing instruments, and the large repeater watch which was presented to him by the staff of St George's before the quarrels, and before the stage when his life was " held in the balance by any who cared to anger him."

An account of the influence of John Hunter on the development of orthopaedic surgery is included in the Historical Reviews of this number of the Journal, but it is impossible within the compass of a few pages to include all the dramatic features of the life of this great man. Why was his marriage delayed ? Was it no more than a financial problem, or was it in consequence of his experimental auto-innoculation with the gonococcus—a study complicated by unexpected infection with the spirochaetes of syphilis ? What was his relationship with his brother, William Hunter ? Was it represented accurately by the letter in which he wrote: " Dear Brother, to-morrow morning at eight o'clock and at St James' Church I enter into the Holy State of Matrimony. As that is a ceremony which you are not particularly fond of, I will not make a point of having your company there " ? What was the attitude of his wife—" a lady noted for her beauty, wit and accomplishment—a social figure in the world of art " when her husband, at his own expense, sent a young man to Greenland to bring back a whale, and continued to add to his ever-growing collection so that " the family were literally forced into the street " ? What was the reason for the burning of Hunter's manuscripts after his death by his brother-in-law, Everard Home ? Was it strictly in accord with Hunter's last instructions, or was it the result of piety, jealousy, or plagiarism ? The reader is referred to the vast volume of accumulated literature which breathes through every page the spirit of an individualist and a great pioneer.

DESTRUCTION BY FIRE OF THE ROBERT JONES AND AGNES HUNT ORTHOPAEDIC HOSPITAL, OSWESTRY, SHROPSHIRE

In the last number of this Journal, almost at the moment of publication, certain pages were put through the press a second time in order to include a stop-press account of the disastrous fire which destroyed the greater part of the Robert Jones and Agnes Hunt Orthopaedic Hospital on Tuesday, January 27, 1948. On a dark and raining night a fire started in the Dispensary. There was a west wind, and the flames spread with alarming rapidity along the communicating corridor, destroying every open-air ward in turn. Six hundred patients, most of them immobilised in splints, frames, and plasters, were pushed to positions of safety and protected, so far as was possible by coats and mackintoshes, from the deluge of rain. All six hundred beds and trolleys were lifted by hand over water hoses, debris, and other obstacles, and pushed one mile along the main road by nurses and sisters who proved expert in their unaccustomed task. There was no single casualty. The greatest personal disaster was that the dentures of two patients were interchanged, thus leading to much confusion ! Fire brigades, assembling from many quarters, found it impossible to control so widespread a series of fires. In the face of the wind the last of the scattered but intercommunicating buildings was destroyed. At about this moment the wind veered round into precisely the opposite direction. Thereupon the fire spread eastward along the main corridor and threatened the remaining wards, the operating theatres, radiographic department, and nurses' home. To the credit of the resident medical staff, which includes surgeons not only from England and Wales but from Australia, Canada, and the United States, a new form of manipulative technique was

applied. A ten-yard gap was torn in the structure of the main corridor. Slates, felts, and beams were avulsed by hand in the face of advancing flames. Spread of the fire was arrested. No operation was ever more successful. In consequence of their efforts enough was saved to restore the functions of this great hospital within five days. It is true that the number of beds was reduced. It is true that for a long period there was no hot water and no light. But routine duties were restored; operations continued; surgeons scrubbed up in cold water; patients were fed—not from the dietetic department which exists no longer but from army tent kitchens set up in neighbouring fields. The destroyed sites

Middle of the night.

Next morning.

are cleared already. A rebuilding committee started work the next day. The shape of new wards, physiotherapy department, administrative block, and concert hall can now be determined. No thought of an Appeal Fund was entertained but within twenty-four hours the flow of bank-notes, cheques, postal orders, and other donations from former patients and supporters, ranging from many hundred pounds to one or two shillings, was so great that a rebuilding account has been opened by John Menzies, Secretary-Superintendent of the hospital. The only comment made by Dame Agnes Hunt, who at the time was a patient herself, was: "I never wanted too big a hospital." Her spirit and the spirit of Robert Jones survives; indeed it has gained new impetus.

SURGEONS' CONFERENCE OF THE MINERS' WELFARE COMMISSION, 1947

Redevelopment of Muscle Function

Mr Nicoll (Mansfield) introducing a discussion on the redevelopment of muscle function after injury said that there were four important properties of muscle, each of which had particular significance: i) power of contraction; ii) velocity or speed of contraction; iii) co-ordination; iv) endurance.

1. Muscle power—This was defined as the equivalent of horse-power in an engine, or what was popularly known as strength. It was measurable and always diminished during immobilisation in plaster whether the muscle was damaged or not, and whether exercises in plaster were carried out or not. Exercises certainly minimised falling off in muscle power, but they never abolished it. There were certain physiological facts which had a bearing on treatment. A muscle graduated its power output according to the load. According to Sherrington's law of muscle action, each individual unit contracted to its fullest extent, or not at all, and therefore the process of adapting power-output to load could be achieved only by varying the number of contractile units brought into play at any given moment. The accurate method of measuring muscle power was to measure the maximum load against which the muscle was capable of working. Another physiological fact of importance was that the efficiency of a muscle fibre varied with the extent to which it was stretched, so that it became mechanically less efficient as it shortened. This had a bearing on the technique of resisted exercises.

Most Rehabilitation Centres now employed resisted exercises in order to build up muscle power There had been criticism of this technique, so that it was appropriate to review the principles on which it was based. In the first place there was the analogy with unstriped muscle which always hypertrophied, providing that the resistance against which it worked was increased gradually. In prostatic obstruction, chronic intestinal obstruction, and high blood-pressure, enormous hypertrophy occurred in the bladder, bowel, and left ventricle respectively. The two factors concerned were the gradual nature of the obstruction, and the fact that the muscle was endeavouring to overcome it almost continuously. On the other hand, if obstruction was acute, then instead of hypertrophy there was passive stretching with atrophy. Secondly, there were the well-known occupational examples of dock-porters developing enormous shoulder muscles, and footballers developing hypertrophy of the quadriceps and calf muscles. In other words, muscle hypertrophy was a response to unusual demands over a long period. In America they had carried resistance exercises a stage further and now preached that high resistance exercises develop true power whereas low resistance exercises develop the property of endurance, which was quite a different thing.

He claimed that there were sound reasons for the adoption of graduated resistance exercises in building up both power and endurance in muscles. When these exercises had caused ill-effects it was not because the principle was wrong, but because the technique had been faulty. Because morphine in excessive doses killed people, or even in normal doses occasionally killed very feeble people, there was no reason for prohibiting the use of morphine in medicine. The apparatus originally developed at the Miners' Rehabilitation Centre at Berry Hill for graduated resistance exercises was cumbersome and mechanically inefficient. A new type of apparatus had now been developed at the Oakmere Hall Miners' Rehabilitation Centre which was better in all respects.

2. Velocity or speed of contraction—This was a property of muscle, partly inherent, and partly acquired by training. The highest speeds of contraction were observed in insects; the wings of certain insects moving so rapidly that they created sound waves. Although the muscles of human beings operated at a much slower speed, much improvement could be achieved by training. The virtuoso pianist was at least two species removed from the rest of us (including the virtuoso surgeon) in this matter of speed. Mr Nicoll was ignorant of the physiological basis of this property, but presumed that it had something to do with the speed of nerve impulse and possibly with lowered resistance at synapses through which it passed. It was certainly amenable to training, the mechanism of which, like memory, would seem to be related to facilitation at the synapses. The importance of this property, apart from enabling people like Horowitz to play piano concertos, was that it was the basis of agility, which was important to miners who had to be prepared to move quickly in certain circumstances. Agility, together with co-ordination, was the basis of dexterity and "timing" which was just as important in work as in sport. Finally, the speed at which a muscle like the vastus internus could be brought into play by reflex action was important. If the medial collateral ligament was stretched, and the stretch reflex travelled quickly, the protective action of the muscle was exerted before any damage was done and this was often a matter of split seconds. It was, therefore, an important property to redevelop in muscles, particularly those like the vastus internus which maintained protective action on a joint. It was a property that had to be redeveloped by training.

3. Co-ordination—The perfect example of co-ordination was in the ballet dancer. Its physiological basis was related to the synergic and antagonistic action of a muscle, as opposed to its function as a prime mover. It was therefore dependent on preserving the sensory mechanisms in muscles and ligaments, and keeping these mechanisms in training by synergic exercises during immobilisation in plaster. The importance

of co-ordination was that it played a great part in what was known as "timing." The skilful miner, by proper timing, could load a ton of coal on to the conveyor belt with a minimum output of energy, just as the expert golfer could drive a golf ball 350 yards down the middle of the fairway with no apparent effort. Like velocity of contraction, this property of co-ordination was responsive to training, and like all such human properties it disappeared rapidly when it fell into disuse. Exercises in plaster were, therefore, of paramount importance as were general exercises for the recumbent patient. These kept the proprioceptive mechanisms and all the complicated reflexes in training.

4. Endurance—Physical endurance was defined as the ability of muscles to go on working at high efficiency over a prolonged period. It was another quality fast disappearing in the human race. People who complained, usually from armchairs, that the miners of to-day could not work half as hard as their grandfathers, were apt to forget that grandfathers were not half the men great-great-grandfathers were. In fact, we were approaching that stage of human evolution depicted by H. G. Wells' "Man in the Moon" who was capable only of sitting in a chair while his enormous head was sprayed with water to keep his brain cool. Two thousand four hundred and thirty-seven years ago, a Greek soldier named Pheidippides, hearing that his country was about to be invaded by the Persians, travelled for two days and two nights, climbing mountains and swimming rivers, to seek aid from the Spartans. His mission having failed, he returned to fight throughout the day in the great Battle of Marathon, in which the Greeks were victorious. He then ran 26 miles 385 yards to Athens, and having delivered the great news to the city elders, dropped dead at their feet. Such feats of endurance are beyond human compass to-day, though there are authenticated examples within recent memory of native runners covering a distance equal to that from London to Manchester in forty-eight hours. The running footmen of the Middle Ages, who ran ahead of their lords to arrange board and lodging (an occupation that might well be revived to-day), also performed prodigious feats of endurance.

Endurance was quite separate from power, which depended on the actual number of functioning contractile units in the muscle—in other words, bulk. Endurance, on the other hand, was often associated with lean spare types like Sydney Wooderson, and it was physiologically an expression of respiratory and circulatory efficiency in muscle—the ability to get rid of waste products fast enough to prevent the onset of fatigue. It was not power, mobility, or even residual deformity that prevented spinal cases from going back to full work—it was the fact that they could only work at normal pace for four hours instead of eight hours; so it was worth while trying to find out how to restore this vital property of endurance.

Mr Nicoll had visited France last year to study methods of rehabilitating injured miners. Next time it might be more profitable to study how they trained racehorses! Whatever the secret, it seemed obvious that the orthopaedic surgeon who was bent on improving the respiratory efficiency of muscles should not lose sight of the fact that there was also a central respiratory mechanism. Breathing exercises should form part of the treatment of every case. The "high repetition, low resistance" technique of the Americans was worthy of trial, but it was far from proved.

Mr A. O. Parker said that muscles varied considerably in their response. His experience in poliomyelitis had been that only certain muscles were favourable for transplantation operations; others gave disappointing results however hard one tried. He had noticed surprising hypertrophy in the shoulder and arm muscles of patients compelled to become dependent on crutches—and this was particularly so in children. Hypertrophy in response to extra demand was, in fact, almost constant in the growing muscle but he wondered if the same applied to adults.

Mr C. H. Cullen referred to the fact that some muscles wasted much too quickly for it to be a simple phenomenon of disuse. In injuries of the medial collateral ligament of the knee, for example, wasting of vastus internus was very rapid indeed and it was a matter of urgency to get back the use of the muscle quickly. He agreed that much could be achieved by training and concentration in the matter of individual muscle development. One of his staff was an expert in muscle control and would demonstrate later. He stressed the importance of not overloading the muscle in resistance exercises and referred to Professor Le Gros Clark's findings that if only the muscle sheath and a few nuclei were left the muscle fibre could regenerate.

Mr Leslie Morris doubted if resistance exercises were necessary. He operated on many professional footballers suffering from cartilage lesions and his usual procedure was to put them back in the second team after five weeks. He never prescribed resisted exercises but his patients seemed to get fit and usually played first-class football within three months.

Sir Thomas Fairbank referred to the time-honoured use of massage and heat before and after exercise periods in those patients who developed pain due to early fatigue. These agents stimulated intra-muscular circulation and lymph flow.

Mr F. G. St Clair Strange said that under normal conditions there must be some mechanism for inhibiting the full output of muscle power. This restraining mechanism was released in conditions like acute mania

in which normally weak and puny people suddenly became possessed of superhuman strength. He had seen the same thing happen during an experiment on hypnotism at which he had been present.

Mr E. A. Nicoll, in reply, said that hypertrophy in response to extra demand was commonly observed in adults as well as children. This was particularly borne out in paraplegics, and one had only to visit Stoke Mandeville to be convinced of it. In reply to Mr Morris, he agreed that some patients got fit if they were left to their own devices, but only if they were normally active and determined types. In such cases, analysis of progressive activities revealed that they were, in fact, subjecting muscles to progressive resistance, though without special apparatus. The principle was unaffected, and what the surgeon had to do was to apply the principle scientifically and methodically so that apathetic types would get better as well as others. He agreed with Sir Thomas Fairbank's views on massage and heat therapy. These agents acted by stimulating the circulatory mechanism in muscle, and it was reasonable to suppose that this mechanism improved by being exercised just as did the muscle itself.

Demonstration—The discussion was followed by a practical demonstration of resisted exercise technique in which the special apparatus developed at Oakmere Hall was demonstrated. Mr James Evans, of the remedial staff, concluded by giving an exhibition of muscle work in which he demonstrated power (weight-lifting), individual muscle control, balancing, and co-ordination. This exhibition was conducted with flawless technique and a fine sense of showmanship and was much appreciated by the audience.

SOUTH-WEST ORTHOPAEDIC CLUB

The first regional meeting of orthopaedic surgeons working in the South-West of England was held at Mount Gold Hospital, Plymouth, on March 6, 1948, with Mr Charles Kennedy of Plymouth in the chair. At a preliminary business meeting it was decided to adopt the name " South-West Orthopaedic Club " in order to indicate the friendly and informal atmosphere which it was hoped would be the keynote of the meetings. Mr F. C. Durbin of Exeter was elected honorary secretary. Four short papers were read.

Recent Developments in the Treatment of Rheumatoid Arthritis—*Mr John Bastow* (Bath) summarised recent important advances, and emphasised the value of biopsy both in diagnosing true rheumatoid arthritis, and in estimating the degree of muscle atrophy before arthroplasty or other operative procedures were contemplated. He said that calcium-gold salts were often used and that the danger of unpleasant reactions was minimised by " anti-lewisite." The transfusion of blood of pregnant women appeared to be of some assistance but there was difficulty in obtaining it. Joint lavage helped by clearing out fibrinous debris.

Arthrography in Congenital Dislocation of the Hip Joint—*Mr F. C. Durbin* (Exeter) recalled the distinction drawn by Jacques Leveuf between true congenital dislocation, and primary subluxation of the hip joint. Mr Durbin showed arthrograms demonstrating this difference, and showed slides illustrating the late subluxation to be expected in primary subluxations if a shelf operation was not performed.

Arthrography as a Diagnostic Aid in Knee Conditions—*Mr A. E. Jowett* (Taunton) used a radio-opaque substance, " pylumbrin." He injected 10 c.cs. of 35 per cent. solution into the knee joint to demonstrate the shape and form of the synovial cavity in various disorders. A very interesting series of slides was shown. Pylumbrin was quickly absorbed from the knee joint; most of it disappeared within thirty minutes of injection and it caused little or no reaction.

Results of Intra-articular Injection of Lactic Acid for Osteoarthritis—*Mr H. Salz* (Plymouth) reviewed the results of lactic acid injection in a small series of sixteen cases. In gross osteoarthritis it did not appear to be of much benefit. In the knee joint, when changes were not advanced, good results were obtained. Maximum benefit was usually produced by the second injection. Sometimes circulatory collapse as well as severe pain followed an injection into the hip joint.

Clinical Demonstration—Clinical cases were demonstrated at Mount Gold Hospital by *Mr Lillie* and the resident medical officers.

JOINT MEETING OF LIVERPOOL MEDICAL INSTITUTION AND MANCHESTER MEDICAL SOCIETY, MARCH 4, 1948

The Backache-Sciatica Syndrome and the Intervertebral Disc—*Sir Harry Platt* recalled Goldthwait's original case, operated on by Harvey Cushing, and said that Goldthwait's first conception of rupture of the annulus fibrosus leading to posterior protrusion of the disc substance with pressure symptoms still held good. A warm tribute was paid to the pioneer work of the Boston Medical School, especially of Mixter and Barr, whose original paper had become classical. He did not consider that spinal fusion as well as removal of the disc was necessary, provided that there was not a long history of backache extending over many years, and that the occupation of the patient was not arduous. Prolapsed disc was the commonest *intra-spinal* cause of sciatica, but he deplored the tendency to believe that all sciatica was due to prolapse

of a disc, the clinical picture of which was characteristic and unmistakable. *Professor Geoffrey Jefferson* said that neuritis could not be dismissed as non-existent, and might be the cause of a single attack of sciatica; approximately one-third of these cases cleared up completely with conservative treatment. He discussed the question as to whether a normal disc prolapsed as the result of trauma, or whether trivial injury caused prolapse of a disc already softened by disease. The disc could be displaced laterally and anteriorly as well as posteriorly into the spinal canal and it was necessary to be sure that there was not impingement of part of the disc on the nerve root in the foramen. The results of operation were most satisfactory, over 80 per cent. of his patients subsequently being able to do a full day's work. *Professor T. P. McMurray* urged caution in arriving at assessment of the final value of removing herniated discs. *Mr A. Sutcliffe Kerr* supported hemilaminectomy as the method of choice. *Mr Bryan McFarland* declared that many patients with " disc syndromes " were cured by complete rest followed by a suspension plaster. *Mr Charnley* warmly supported radical measures. *Dr Smiley* advised full investigation and preliminary conservative treatment before operation. *Dr Lennox Johnston* recorded a personal experience of cure by manipulation. *Mr Sayle Creer* advised operation.

HUDDERSFIELD MEDICAL SOCIETY

The Huddersfield Medical Society is one of the oldest medical societies in England. It has been continuously in session, and in the winter months has held regular meetings, every year since 1813. On February 11, 1948, *Mr Bryan McFarland* (Liverpool) showed cinematographic records of interesting and unusual orthopaedic cases. Lively discussion followed on the problems of lipodystrophy, Legg-Perthes' disease of the hip joint, congenital club foot, and the treatment of destructive gap fractures of long bones.

BRITISH MEDICAL ASSOCIATION—116th ANNUAL MEETING, JUNE 1948

The 116th annual representative meeting of the British Medical Association will be held in Cambridge from June 25 to July 2, 1948. The orthopaedic section will meet under the presidency of Professor T. P. McMurray (Liverpool). The vice-presidents will be Mr N. Ross Smith (Bournemouth), Mr R. W. Butler (Cambridge), and Mr H. A. Brittain (Norwich); the honorary secretaries are Mr Bourdillon of Addenbrokes Hospital, Cambridge, and Mr H. Langston of Gifford House, St Giles Hill, Winchester. The programme will include a discussion on the clinical significance and treatment of lesions of the intervertebral discs, opened by Professor Geoffrey Jefferson (Manchester) and Mr Norman Capener (Exeter); a discussion on the operative treatment of fractures; and papers on the injection treatment of osteoarthritis by Mr Grant Waugh (Sunderland), and on the treatment of osteomyelitis.

Discussions and papers in other sections will include: Recent work on Proteins by Professor A. C. Chibnall, Dr J. A. V. Butler, Dr Leonard Colebrook, and Professor G. Pickering; Plasma and Blood Derivatives in the Treatment of Burns by Professor E. C. Dodds; Physiological Basis of Neuromuscular Disorders by Sir Henry Dale and Dr W. S. Feldberg; Acetylcholine and Neuromuscular Transmission by Dr Bernard Katy; Excitation at the Myoneural Junction by Dr Andrew Wilson; and Poliomyelitis by Dr W. Gunn and Dr F. W. Bunting.

INTERNATIONAL CONGRESS ON INDUSTRIAL MEDICINE, LONDON, SEPTEMBER 1948

The ninth International Congress on Industrial Medicine will be held at Caxton Hall, London, from September 13 to 17, 1948, under the patronage of Their Majesties the King and Queen. The Vice-patrons include the Right Honourable Mr Ernest Bevin (Secretary of State for Foreign Affairs), Mr Isaacs (Minister of Labour), Mr Aneurin Bevan (Minister of Health), Mr James Griffiths (Minister of National Insurance), Mr Strauss (Minister of Supply), and Mr Gaitskill (Minister of Fuel and Power). The presidents of the Royal College of Physicians and the Royal College of Surgeons will serve as presidents of the Congress. The chairman of the Planning Committee is Dr Donald Hunter (London Hospital) and the chairman of the British Organising Council is Mr T. E. Stowell (London). The discussions will include biological, psychological, physical, chemical, and architectural aspects of environment in industry; organisation of industrial medical services; surgical aspects of industrial medicine; chemical and thermal burns; medico-legal aspects of industrial hazards; and rehabilitation after industrial injury. The MacKenzie Industrial Health Lecture will be given by Mr H. E. Griffiths on Rehabilitation in Industry. Visits will be arranged to the London Hospital, Industrial Health Exhibition of the Royal College of Nursing, Queen Elizabeth's Training College for the Disabled, Roffey Park Rehabilitation Centre, Ministry of Labour Rehabilitation Centre, Egham, Birmingham Accident Hospital, Austin Motors Rehabilitation Centre, Mersey Docks and Harbour Board Medical Centre, Liverpool. Arrangements can be made with Miss Mawdesley, Organising Secretary, B.M.A. House, Tavistock Square, London, W.C. 1.

AN ARTIST'S VIEW OF THE ORTHOPAEDIC SURGEON'S THEATRE

An exhibition of drawings by the well-known sculptor, Barbara Hepworth, which was of particular appeal to orthopaedic surgeons, was held last month at the Lefevre Galleries in London. In former days many of the old masters found interest in the work of medical men. A well-known example is Rembrandt's "The Anatomy Lesson." Most surgeons are familiar with the work of medical artists who brave the rigors

"Median" by Barbara Hepworth
(Peripheral nerve injury operation)

"Reconstruction" by Barbara Hepworth
(Arthroplasty of the hip)

of the operating theatre to record or illustrate pathological detail and operative technique. In Barbara Hepworth we have an artist of a different type with both stamina and vision, who sought to appraise, not technical detail, but the sincerity, harmony, rhythm, tenderness, and simple drama of the operating theatre. Little, perhaps, do surgeons realise the classic beauty of their surroundings—a beauty based upon perfect architectural conditions designed for a purpose, and a grouping which appeals to the sculptor because of the associated movement of individuals and objects, with perfect co-ordination, towards one

purpose within a central stage. These drawings seem to reveal the spirit of surgery—the spirit of inquiry, intensity of proper solicitude, power of the craftsman, unhurried activity, and energetic poise. For the layman the artist has distilled out of the picture the sentimental and the morbid. In these drawings there is no individual portrayal, but there is an uncanny sense of the unseen; indeed the sense of the good surgeon himself—always conscious of the unseen " person " beneath his hands and never callous of his " material." Surgeons may or may not feel flattered by these drawings, but they cannot fail to be impressed with their beauty and by the sincerity of the artist. N. C.

EMPIRE RHEUMATISM COUNCIL

The spring week-end course of the Empire Rheumatism Council was held in Apothecaries' Hall, Blackfriars Lane, London, in March 1948. Lectures were given by Dr Kersley on Gout, Dr Fletcher on Osteoarthritis, Dr Tegner on Spondylitis, Dr Bonham-Carter on Still's Disease, Dr Copeman on Sciatica, Dr Savage on Rheumatoid Arthritis, and Mr W. D. Coltart on Orthopaedic Aspects of Rheumatic Diseases.

NORTHERN IRELAND

THE NORTHERN IRELAND ORTHOPAEDIC SCHEME

It was an historic day for Northern Ireland in 1940 when Sir David Lindsay Keir, Vice-Chancellor of Queen's University, Belfast, called a meeting of representatives of the hospitals and other statutory and voluntary bodies throughout the six counties. The Northern Ireland Council for Orthopaedic Development was established, under the presidency of the Duchess of Abercorn, with the object of developing an orthopaedic scheme for the Province. Sir David, who was elected Chairman of the Council, carried out the wishes of Trustees of the Nuffield Fund for Cripples, who in 1937 had set apart £26,000 for the development of orthopaedics in Northern Ireland, and the Central Council for the Care of Cripples which acted in advisory capacity.

Northern Ireland, comprising the counties of Antrim, Armagh, Down, Fermanagh, Londonderry and Tyrone, with the County Boroughs of Belfast and Londonderry, is peculiarly suited to the development of centralised medical schemes, and this has been recognised by the Parliament of Northern Ireland which passed a Tuberculosis Act in 1946, centralising the scheme for all forms of tuberculosis. Northern Ireland's population of 1,348,000 is nearly one-third that of Ireland as a whole. Belfast, its chief industrial centre, has a population of rather less than half a million, with its University admitting approximately 120 medical students each year. The main industry in the rest of the Province is agriculture. From the point of view of economics and social services, Northern Ireland is on a par with Great Britain, constitutionally being an integral part of the United Kingdom with a Parliament of its own, federal in type, but with certain powers reserved to the United Kingdom Parliament. On the whole the people are a mixture of Celt and Scot, and many retain the qualities of their Scottish ancestry—hard-working, industrious and thrifty, slow to accept new ideas, but determined in their pursuit of ideas once they have been proved worth while.

Thus in 1941 the Northern Ireland Council for Orthopaedic Development launched its Orthopaedic Scheme. In consequence of the war, the Council was unable to carry out its first objective, namely the establishment of an open-air Orthopaedic Hospital. Instead it set out to provide clinical facilities at the main centres of population throughout the Province. A panel of surgeons was formed, and Sister Gwladys Morris came from the Robert Jones and Agnes Hunt Orthopaedic Hospital, Oswestry, to do her magnificent pioneering work.

From small beginnings at Coleraine and Londonderry the scheme progressed until now there are eleven clinics under the Council's auspices with a total of more than 3000 patients. These clinics are situated at Londonderry, Coleraine, Ballymoney, Ballymena, Ballycastle, Downpatrick, Omagh and Armagh, and in three Belfast Hospitals. Further extension is planned on a regional basis so that the whole Province may have access to orthopaedic clinical facilities. In addition, the Council has sought to make arrangements for hospital treatment for its patients. The lack of its own open-air hospital has proved a source of difficulty and delay in arranging effective treatment. However, beds have been made available at various Belfast Hospitals and in particular at the Ministry of Health Emergency Hospital. Here the Council's visiting orthopaedic surgeons are in charge of their own cases referred from the clinics, and thus the principle of continuity of treatment is maintained. Every effort has been made towards the establishment of an open-air hospital; plans were drawn up, sites were investigated and the cost was assessed; but the Council was asked not to put the plans into action because the hospital will soon be considered by the Hospitals Authority to be appointed under the new Health Services Act, in relation to the complete needs of hospitalisation in the Province.

The Council has four visiting surgeons—Mr H. P. Hall, Mr Maurice Lavery, Mr Norman Martin, and Mr R. J. W. Withers; and three orthopaedic physiotherapists—Miss G. M. Morris (After-Care Superintendent), Miss M. Fearing, and Miss S. Brown.

The Vice-Chairman of the Council is Mr S. T. Irwin, C.B.E. Under the Executive Committee administration, execution, and organisation of work is carried out by Mrs Doreen McConnell, organising secretary to the Council, with an office staff of three. Voluntary Committees have been set up in various areas, and members of the Voluntary Aid Detachment give their services in the clinics as part of the orthopaedic team.

In aiming to provide a complete orthopaedic scheme, the Council has interested itself in all aspects of the care and cure of cripples: special education, vocational training, rehabilitation and general welfare. A continuous effort has been made to acquaint the general public with the need for orthopaedic treatment by which to prevent and cure crippling diseases and defects. Talks have been given and films are shown all over the country.

Noteworthy events have been the recent acceptance of the Presidency of the Council by Her Excellency the Countess Granville, D.C.V.O., wife of the present Governor of Northern Ireland; the incorporation of the Council; the training at the Wingfield-Morris Orthopaedic Hospital of Mr Norman Martin, now one of the Council's visiting orthopaedic surgeons; and the servicing at Purdysburn Fever Hospital of the poliomyelitis cases during the 1947 epidemic.

The Northern Ireland Council owes much to many individuals in Northern Ireland; to the Trustees of the Nuffield Fund for Cripples and the Central Council for the Care of Cripples; and to Sir Reginald Watson-Jones, Mr Norman Capener, Mr G. R. Girdlestone, and Professor Mackintosh, who have visited Northern Ireland with the purpose of developing their work.

AUSTRALIA

The next meeting of the Australian Orthopaedic Association will be held in Perth on August 11–12, 1948. This will immediately precede the meeting of the Australasian Medical Congress, organised under the direction of the British Medical Association, which meets every third year. This is the first occasion that the Congress has been held in Perth. The meeting embraces every section of medicine and surgery and there will be a section of orthopaedics under the presidency of Mr Charles Littlejohn of Melbourne. The Secretary of the orthopaedic section is Dr A. L. Dawkings of Perth.

UNION OF SOUTH AFRICA

In South Africa the importance of orthopaedic surgery has been recognised for many years in the University cities of Johannesburg and Cape Town. This arrangement of two isolated centres of orthopaedic service continued until comparatively recently when the generous grant from Lord Nuffield stimulated the development of orthopaedic centres in many other cities.

In the University of the Witwatersrand in Johannesburg the responsibility for undergraduate and post-graduate teaching rested on Mr Fouché, who was the first lecturer in orthopaedic surgery. Undergraduate instruction consisted in the delivery of a course of lectures, extending over two University terms. The student was also obliged to attend clinical demonstrations in the Out-Patient Departments of the General and Children's Hospitals. For the post-graduate who wished to obtain the Mastership of Surgery with specialisation in orthopaedics, a two years course, both theoretical and practical, was obligatory. During the past year Mr Fouché has retired and the University has appointed Mr Edelstein to take over the control of teaching.

Master of Surgery in Orthopaedics, Johannesburg—Professor T. P. McMurray (Liverpool) writes: "My experience of the Witwatersrand University was gained through invitation to serve this year as external examiner in orthopaedic surgery for the degree of Master of Surgery. I found that the instruction, and the experience gained by candidates for the examination, was in every way excellent. During the two years of training, the student receives continuous instruction in general surgery from the Professor of Surgery, and in general and special pathology from the Professor of Pathology. At the same time he is appointed as clinical assistant to Mr Edelstein, Mr Polonsky, Mr du Toit, or Mr Moller. In these posts he has the opportunity of dealing with large numbers of patients, not only in the out-patient department but also in the wards and operating theatre. These unusual opportunities for training are possible only on account of the immense amount of clinical material which is available in the orthopaedic services. The clinics include large orthopaedic and fracture departments of the general hospital, and the even larger department in the Non-European hospital, together with the orthopaedic service of the Children's hospital.

"In common with the practice at most large hospitals in Britain fracture clinics are held daily, and the work is divided evenly between members of the orthopaedic staff of the hospital. Patients requiring long periods of hospital treatment for tuberculous arthritis, infantile paralysis, or congenital deformities, are treated in the Hope Hospital which is run under the direction of Mr Edelstein, and has the advantages of high altitude, and of the enthusiasm of an active staff of nurses and physiotherapists.

"The standard of examination for the Mastership of Surgery is very high. The tests consist of written sections on general surgery, general and special pathology, and orthopaedic surgery, together with a

searching clinical examination. The University has rightly decided that knowledge of part of surgery alone is not sufficient to qualify the candidate for a Master's degree. It insists upon a satisfactory knowledge of all branches of surgery and surgical pathology. The tests in general surgery were in the hands of Professor Underwood and Professor Brebner. The pathological examination was held in a private ward of the General Hospital where Professor Strahan did not allow his powers of examination to be impaired by the fact that, at that time, he was a patient himself. No section of general surgery or pathology was left untouched. The marking was fair but firm. As the result of an examination extending over seven days, the pass rate was only 25 per cent."

Orthopaedic Surgery in Cape Town—In Cape Town Mr Hamilton Bell holds the posts of Lecturer on Orthopaedic Surgery at the University, and senior orthopaedic surgeon to the Groot Schur General Hospital. University teaching has been confined to undergraduates, and although the University holds examinations for the degree of Master of Surgery, a special examination in orthopaedics has not yet been held. Holding the post of assistant surgeon at the Groot Schur Hospital, and in charge of the active orthopaedic research department in the University, is Mr A. J. Helfet, who is also a clinical teacher in orthopaedic surgery.

Long-stay patients are accommodated in the Lady Michaelis Home, which has been specially designed and equipped for this purpose. The Home, which is under the direction of Mr Bell, deals largely with children suffering from infantile paralysis, tuberculous arthritis, and congenital deformities. In addition to physical rehabilitation the patients receive school instruction up to the matriculation standard.

The undergraduate student receives adequate and concentrated instruction during the whole of one University term through a planned course of lectures and practical training in the orthopaedic out-patient department of the General Hospital. Further attendance at the out-patient clinics is voluntary, but this privilege is taken advantage of by a considerable proportion of the students. As in Johannesburg, undergraduate instruction in orthopaedic surgery is adequate, including out-patient, ward and operative teaching, combined with training in orthopaedic pathology. Facilities for full post-graduate training in orthopaedic surgery are available in Cape Town, but at present it is considered that the number of trained orthopaedic surgeons is sufficient for the posts available in the Union.

Pretoria, Durban, and Port Elizabeth now have active orthopaedic centres under Mr du Toit, Mr Allen, and Mr Maister. The Orthopaedic Surgeons' Group of the Union of South Africa, which is an active clinical body, meets in the different centres twice each year.

Poliomyelitis in South Africa—We learn with regret that South Africa is in the midst of a severe epidemic of poliomyelitis. Since January of this year, 1062 cases have been reported (Europeans 784, non-Europeans 278). Cases have been reported in Johannesburg 535, the Transvaal Province 832, Cape 82, Orange Free State 56, and Natal 96. The epidemic is notable for the high proportion of adult cases.

The Orthopaedic Section of the Thirty-sixth South African Medical Congress will meet in Pretoria in July 1948.

COLONIES OF EAST AND WEST AFRICA

Panel of Medical Consultants for the Colonies—The Secretary of State for the Colonies has recently appointed a panel of medical visitors to East and West Africa (*Times*, March 8, 1948). The scheme was proposed by Professor H. J. Seddon, Nuffield Professor of Orthopaedics at Oxford, and the late Dr W. H. Kauntze, Medical Adviser to the Secretary of State. The Nuffield Foundation has generously undertaken to finance the scheme during an experimental period of six years.

The Colonial Medical Service labours under several peculiar difficulties, one of which is the discouragement which arises from the isolation of many of its members. There is lack of professional companionship and of contact with leaders in the various fields of medical activity. During the war, medical consultants in the Army Commands of East and West Africa, mostly men from British teaching hospitals and medical schools, were strikingly successful in stimulating the enthusiasm of junior colleagues, many of whom were stationed in remote places. The Colonial Office has followed this precedent in undertaking a development of Colonial welfare which promises to be of great significance.

Specialists from a panel of eighteen will each visit Africa twice in the next six years. If the experiment is successful it may be extended to other parts of the Colonial Empire. The consultants will go out with the blessing of the Secretary of State, but they will also be under the aegis of the Nuffield Foundation, and will have no departmental or official status. It is believed that men in the field will discuss their difficulties with a freedom which would probably have been difficult or impossible if the consultants had been government servants. The various specialists, most of whom are well-known teachers, have been chosen for their particular knowledge of the special problems of colonial medicine such as tropical diseases, tuberculosis, venereal disease, and child health; they will be able to give valuable advice to administrators, public health workers, and clinicians. But even more the visiting specialists have been chosen for their qualities of congeniality and friendliness which will make them acceptable guests in the bungalows of men who work in solitude.

NEW ZEALAND

ANNUAL MEETING OF THE ROYAL AUSTRALASIAN COLLEGE OF SURGEONS, 1948

The Annual Meeting of the Royal Australasian College of Surgeons was held in Dunedin, New Zealand, on January 28, 1948.

Bilateral Rupture of the Long Tendon of the Biceps—*Mr Kenrick Christie* reviewed the literature and referred to the analysis of Gilcreest which showed that rupture of the biceps tendon involved the long head in 65 per cent. of cases, the short head in 26 per cent., both long and short heads in 7 per cent., and the lower common tendon in 4 per cent. Only in 5 per cent. of cases was the rupture bilateral. Senile degeneration due to diminished blood supply, together with attrition due to the peculiar course of the long tendon over the head of the humerus, which was often the site of arthritis with marginal osteophytes, were the predisposing causes. The exciting cause was flexion of the elbow against powerful resistance.

In long-standing ruptures, and in old age, operation was often not indicated, but in other cases early operation was advised. Mr Christie demonstrated the results of passing the biceps tendon through the tendon of insertion of the pectoralis major tendon, looping it round and suturing it to itself. After operation the limb was immobilised in the flexed position for three weeks. Return to work could be expected in eight to twelve weeks. The prognosis was good. Even in comparatively late cases it was possible to regain almost normal muscle power. Medico-legal aspects were discussed.

NORWAY

Decoration of Dr Smith-Petersen—On December 6, 1947, Dr M. N. Smith-Petersen was decorated by the King of Norway with the Grand Cross of the Royal Order of St Olave—the highest Norwegian award that it is possible to confer.

Lectures in Norway by Mr I. Lawson Dick—During the last few months British Medical Association Lectures have been given by distinguished British physicians and surgeons in Czechoslovakia, Denmark, Spain, and the Netherlands. Mr Lawson Dick took the place of Sir Reginald Watson-Jones in Norway and lectured on orthopaedic surgery. After his return he wrote: "The welcome could not have been more cordial. National feeling in Norway is very strong and they have a high regard and warm affection for Britain. Both in Bergen and in Oslo we had marathon evenings with two lectures, one straight after the other, but the audiences were so responsive that it was no effort. The long-stay hospitals for tuberculosis and orthopaedics are very fine. The Martina Hausen Hospital near Oslo, and the Kysthospitalet i Hagevik, which is on the coast about fifteen miles south of Bergen, are lovely buildings, magnificently situated, in superb surroundings. Medicine in Norway bears the mark of long isolation. Their fracture treatment shows much more of the influence of Bohler than is now seen in Britain. But some of their work is excellent. The imprint of Dr Smith-Petersen's teaching is everywhere recognisable. Specialisation is not yet completely developed but they are taking active steps to introduce it. Five years of occupation did not break the Norwegian courage. Nearly all the doctors had been in concentration camps. The Secretary of the Norwegian Medical Association had been interned for five years because his colleagues would not subscribe to the new German controlled Medical Association. My host in Stavanger had tears in his eyes when he told me how he saw the first British paratroops march down the hill into the town. I shall never forget my visit which was made wonderfully enjoyable by boundless Norwegian hospitality."

SPAIN

In February 1948, the Spanish Ministry of Labour approved the establishment of a School of Industrial Medicine, administered by the Spanish Institute of Medicine and Industrial Safety, and attached to the Central University of Madrid as a post-graduate school. One of the objects is the training of specialists in orthopaedic and traumatic surgery. A University degree in these subjects will be demanded of those filling orthopaedic vacancies in the National Insurance Scheme, and in the Insurance Companies concerned in industrial medicine. Research will be carried out over a wide field of industrial medicine. We welcome this practical understanding of the important part which orthopaedics has to play in industrial medicine.

FIRST INTERNATIONAL POLIOMYELITIS CONFERENCE—NEW YORK

The first world conference on infantile paralysis is to be held in New York on July 12–17, 1948. It is sponsored by the National Foundation for Infantile Paralysis of the United States. There will be representatives from China, Sweden, France, Argentine, Brazil, Great Britain, and other countries. Professor H. J. Seddon will show a film prepared in Oxford.

Book Reviews

SURGICAL TREATMENT OF THE MOTOR-SKELETAL SYSTEM. Edited by Frederic W. BANCROFT, A.B., M.D., F.A.C.S. (Supervising Editor), and Clay Ray MURRAY, M.D., F.A.C.S. (Associate Editor). 10×6½ in. Pp. xix+1254, with 1063 figures. Index. Two volumes. 1946. London: J. B. Lippincott Company. Price £6.

The field of knowledge in every branch of surgery is becoming so expanded that already it is almost impossible for a single author to accomplish the task of writing a comprehensive volume. Inevitably, the text-book of multiple authorship has come to stay. The system has certain disadvantages, the chief of which is that the quality of the various contributions, both in substance and in style, is seldom consistently maintained throughout the book. Another common failing arises from the tendency among some authors—possibly invited to contribute because of original work previously published—to put forward their own particular methods to the exclusion of others, often equally effective and more generally recognised. This book accords with this general statement.

In conception it is admirable—it sets out to tell the surgeon "what to do, when to do it, and how to do it" in relation to the whole field of surgery of the motor-skeletal system. In large measure the editors and forty-one contributors have succeeded in this aim.

The first volume is devoted mainly to the management of deformities, infections, tumours, and other non-traumatic orthopaedic affections. The second volume deals with the treatment of injuries, and includes an admirable discussion on the present position of chemotherapy, and a chapter on war surgery.

With few exceptions the various subjects are covered fully and in an up-to-date manner. Among others, the sections on the general principles of fracture treatment, and on the management of scoliosis, are particularly valuable, though the latter is made difficult to read by the author's complicated style. From the chapter on tuberculosis of bones and joints the reader inevitably gains the impression, perhaps unintended, that a short cut to cure can frequently be achieved by surgical treatment. This is a fallacy which has been recognised in this country since the days of H. O. Thomas and Sir Robert Jones.

In the section dealing with congenital talipes equinovarus it is a little surprising to find that treatment by Denis Browne splints receives no mention. Surely too it is illogical to discuss the surgical treatment of this deformity before considering non-operative forms of treatment. Similarly one is amazed to find that in describing the technique of insertion of a three-flanged nail for fractures of the neck of the femur the contributor makes no mention of the use of a preliminary guide wire and cannulated nail. This omission is rectified by the editor in one of the many interpolations he has made throughout the book whenever he has felt that additional comment was required for completeness.

The production, as is usual with Lippincott publications, is excellent, though there are minor points of criticism: the reproduction of radiographs is in many instances imperfect; misprints are not infrequent; and the legends of two illustrations (Figs. 162 and 163) are reversed. It is a pity too that there is no more clear indication of the number of the volume on the outside cover—the two volumes are distinguishable only by a single star on one and a double star on the other. The inclusion of a complete index of the whole work in each volume is a particularly welcome feature which might be more widely adopted in multi-volume text-books.

This work cannot fail to achieve a wide reputation since it fills a long-felt want for a comprehensive and up-to-date guide to the treatment of orthopaedic conditions. Orthopaedic surgeons throughout the world will regret the tragic death, since the book was published, of the Associate Editor, Clay Ray Murray. His virile and receptive mind would have welcomed criticism and he would have done much to improve subsequent editions of this already invaluable book.—H. OSMOND-CLARKE.

THE CAUSATION AND TREATMENT OF DELAYED UNION IN FRACTURES OF THE LONG BONE. By Kenneth W. STARR, O.B.E., M.S., F.R.C.S., F.A.C.S., F.R.A.C.S., Surgeon, Sydney Military Hospital, Visiting Surgeon, Concord Military Hospital. 9½×6½ in. Pp. xiv+215, with 106 figures, 3 coloured plates, and 4 tables. Index. 1947. London: Butterworth & Co., Ltd. Price £2, 2/–

This book, which is a Jacksonian Prize Essay, is divided into three parts; the first deals with the structure of bone, the chemistry of bone, the cycle of bone reconstruction, and the relationship between structure and function in bone morphology. The second part is concerned with the healing of fractures, and the third with the etiology and treatment of delayed union. Fifty illustrative cases are reported briefly in one of the appendices. Consequently, the problems of bone growth, bone repair and its complications are considered from many aspects.

In his introduction, the author makes a somewhat naïve acknowledgement of his "felony in borrowing

without shame from the scientific riches stored in the bibliography." The text abounds with references, but the reader is sometimes puzzled to know which part is the borrowed riches, and which the author's own opinion. The bibliography contains 615 references but omits any mention of Hertz's work on Vitamin C. The proven deleterious effect of lack of Vitamin C in the healing of fractures is not considered in the text. The book is profusely illustrated. The micro-photographs and radiographs are excellently produced, but many of the tables and diagrams, for example Fig. 63, are reduced to such a degree that a magnifying glass may be required to read them. The text contains a wealth of information, but critical apprisement and correlation of the information is somewhat lacking.—Robert I. STIRLING.

BRACHIAL PLEXUS: LOCAL ANAESTHESIA. By R. R. MACINTOSH, M.A., M.D., F.R.C.S., D.A., Nuffield Professor of Anaesthetics, University of Oxford, William W. MUSHIN, M.B., B.S., D.A., First Assistant Nuffield Department of Anaesthetics. $7\frac{1}{2} \times 5$ in. Pp. 55, with 33 figures. 1944. Oxford: Blackwell Scientific Publications, Ltd. Price 10/6.

FIG. 8. Page 18.

The first edition of this small monograph was published in 1944. In the second edition, which has now appeared, there have been no material additions. The striking features of the book are the excellence of the illustrations and the clarity of presentation.

A short history of the various methods of producing brachial plexus block is followed by a discussion on its indications and advantages. The section dealing with the anatomy of the plexus is profusely illustrated in a way which emphasises the important topographical relations of the brachial plexus in the region of the first rib. Fig. 8 is an example. The actual technique is also illustrated admirably and made abundantly clear. The method described is, with minor modifications, essentially that of Patrick, which is undoubtedly the most reliable. (PATRICK, J.: The Technique of Brachial Plexus Block Anaesthesia. British Journal of Surgery, 1940, **27**, 734.)

References are made to premedication, and local anaesthetic solutions, and the book concludes with a brief description of the anatomy of the stellate ganglion and the signs of stellate ganglion paralysis.—J. D. EBSWORTH.

THE INTERNAL FIXATION OF FRACTURES. By Charles Scott VENABLE, M.D., M.S., F.A.C.S., and Walter Goodloe STUCK, M.D., M.S., F.A.C.S. $10 \times 6\frac{3}{4}$ in. Pp. xi+237, with 84 figures. Index, 1947. Oxford: Blackwell Scientific Publications, Ltd.

Less than fifty years ago the internal fixation of fractures was so unreliable and dangerous a method of treatment that it was condemned by all but a few of the more adventurous pioneers of surgery. Later the method achieved a considerable vogue and in the hands of a few skilled surgeons was attended by satisfactory results in a fair proportion of cases. In general, however, the results continued to be indifferent, and it was not until the introduction of metals which are relatively inert in the body that consistently satisfactory results began to be obtained. With the development of inert metals to their present state the principle of internal fixation of fractures has now achieved an established place in orthopaedic surgery, and it is generally regarded as a safe and reliable procedure provided that certain conditions are observed. Nevertheless the fact that cases are not infrequently seen even to-day where the choice of method or of materials has been unfortunate emphasises the necessity for every surgeon who undertakes the treatment of fractures to make himself thoroughly familiar with recent developments in this field; and this book will serve a useful function in bringing together under one cover essential features of the many contributions on internal fixation.

The book is arranged in two main sections. In the first part the authors sketch historical outlines of the development of the use of metals for internal fixation, leading up to the introduction of the relatively inert stainless steels and of vitallium. These and other metals are described in some detail; their relative advantages and disadvantages are discussed; and the technique of their application is considered. An important point which is wisely stressed is that two metals of different composition, even though each is

individually inert, may nevertheless set up undesirable electrical reactions if used together in the same operation area; all metal parts used in a given area must be identical in composition if this risk is to be avoided.

The second part of the book deals with the application of the principles of internal fixation to various individual fractures of the upper and lower limbs. While the approach is reasonable, and the exposition clear, this section does not appear to fill any significant gap in surgical literature; the survey is too limited in scope to be of use as a comprehensive treatise on fractures—and the special problems of internal fixation which are discussed are for the most part covered fully in modern text-books on the subject. Nevertheless there are some stimulating chapters. In particular the problem of fractures of the neck and trochanteric region of the femur is discussed very adequately. There will be some, however, who will not agree that avascular changes in the femoral head can be avoided by early and adequate reduction and fixation. Others—they may be fewer in number—will require further evidence before they are convinced that internal fixation is the treatment of choice for the trochanteric type of fracture, or that the mortality in this type is in fact less with operative treatment than with the best conservative treatment in a comparable series of cases.

It is surprising that there is no description of the use of the long intra-medullary nail. This method of internal fixation is widely accepted as having a place in fracture treatment, particularly for certain fractures of the shaft of the femur, and some reference to it would have been expected in a work of this kind. Another minor criticism is that the authors advocate, or at least condone, the use of the heavy rubber tube type of tourniquet. Nevertheless in general the principles advocated are sound and the judgment is well balanced. The book should have considerable appeal, particularly to those who are in the course of orthopaedic training and to those who have special interest in the mechanical aspects of fracture surgery.—J. C. Adams.

DISEASES OF THE JOINTS AND RHEUMATISM. By Kenneth Stone, D.M., M.R.C.P., Honorary Physician, British Red Cross Society Clinic for Rheumatism, Peto Place, London. $9\frac{1}{4} \times 6$ in. Pp. x+362, with 58 figures. Index. 1947. London: Heinemann (Medical Books), Ltd. Price 30/–.

This book is intended to remedy defects in the teaching of rheumatology, which the author would prefer to call " Orthopaedic Medicine." It is no longer advisable to stress any cleavage between medicine and surgery, particularly in orthopaedics. One feels sure that this is not Dr Stone's intention; nevertheless, co-operation with an orthopaedic colleague would have produced a more comprehensive and accurate review. Having made this complaint one must add that there is much to commend in this book. Dr Stone does not confine himself to a factual discussion: there are many pleasant excursions into the fields of medical philosophy and history which will stimulate the reader to think.

The first part of the book is devoted to a discussion of general principles and the pathology of arthritis. Very rightly the work of Hilton on physiological rest is emphasised; nevertheless, too little space is devoted to the important matter of physiological splintage. The next six chapters discuss diseases of the joints upon a regional basis. The large problem of the osteoarthritic hip is given very brief discussion. Surgery still plays an important part in treatment and yet the matter is dismissed in eleven or twelve lines. No mention is made of arthrodesis, let alone arthroplasty. The surprising statement is made that " as a rule surgical treatment will never be considered for a patient over fifty-five years of age."

The final section, comprising five chapters, is devoted mainly to the subject of muscular rheumatism and fibrositis. Dr Stone discusses in some detail the anatomy and physiology of the autonomic nervous system, and explores the fascinating hypothesis of Eppinger and Hess on the subject of vagotonia. He regards pronounced parasympathetic stimulation as an explanation of many of the phenomena of muscular rheumatism. The book concludes with a discussion of lumbago and sciatica. On the subject of sciatic scoliosis we read that " text-books still affirm that the erector spinae are in a state of spasm. But a spasm of the erector spinae would *hyperextend* the lumbar spine: and this one never sees." The problem of spinal muscle spasm in these cases is a very real one and deserves fuller and more accurate statement. A great deal of space is devoted to the theories of Cotugno (1764) on nervous sciatica, a discussion which tends to confuse rather than illumine the mind of the reader. In the first attack of sciatica due to intervertebral disc lesions rest in bed is recommended, but " if weeks elapse with no sign of abatement, and if to be up and about is imperative, the only recourse is to immobilise the spine in plaster: a method which in some instances appears to be successful." Thus is dismissed the conservative orthopaedic treatment of this common disability. These occasional deficiencies in the details of orthopaedic practice are unfortunate, for the book contains so much that is good for the general practitioner and senior student.—Norman Capener.

EXCERPTA MEDICA. Edited by M. W. Woerdeman, Professor of Anatomy and Embryology, University of Amsterdam, A. P. H. A. De Kleyn. Professor of Oto-rhino-laryngology, University of Amsterdam, and W. P. C. Zeeman, Professor of Ophthalmology, University of Amsterdam. In fifteen sections. Section IX, Surgery. $9\frac{3}{4} \times 6\frac{1}{2}$ in. Pp. 120. Index of authors. 1947. Edinburgh: E. & S. Livingstone, Ltd. Price £9, 6/– for annual volume of Section IX. Paper cover.

This work is succinctly described as " A Complete Monthly Abstracting Service of the World Medical Literature comprising fifteen sections and covering the whole field of theoretical and clinical medicine." The abstracts are in English, are made by experts, and are signed. In addition to current articles, the more important ones going back to 1940 which have not been readily available on account of the war will be included in the earlier numbers. Each volume will have a subject and authors index. The abstracts in the Surgery section are arranged under seventeen headings of which number 13 is devoted to Orthopaedic Surgery, which conveniently follows Vascular Surgery and precedes Traumatic Surgery. Those interested in the territorial claims of the specialties will note that Orthopaedic Surgery here comprises Physical Medicine, Fractures, Dislocations, Bones, Joints, Muscles, Tendons, Bursae, and Amputations. Traumatic Surgery is confined to Thermal Injuries, Wounds, Shock, and Transfusion. Other headings of interest to the orthopaedic surgeon are 15 and 16 on Military and Experimental Surgery respectively, and 1 to 5 on Anaesthesia, Pre- and Post-operative Therapy, Surgical Technique, Surgical Infections, and Tumours. The abstracts are very well done, and we should be most grateful to our Dutch friends for this important contribution. It is salutary also to remember the large part which the American nation has played towards making English the scientific language of the world.—H. Jackson Burrows.

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